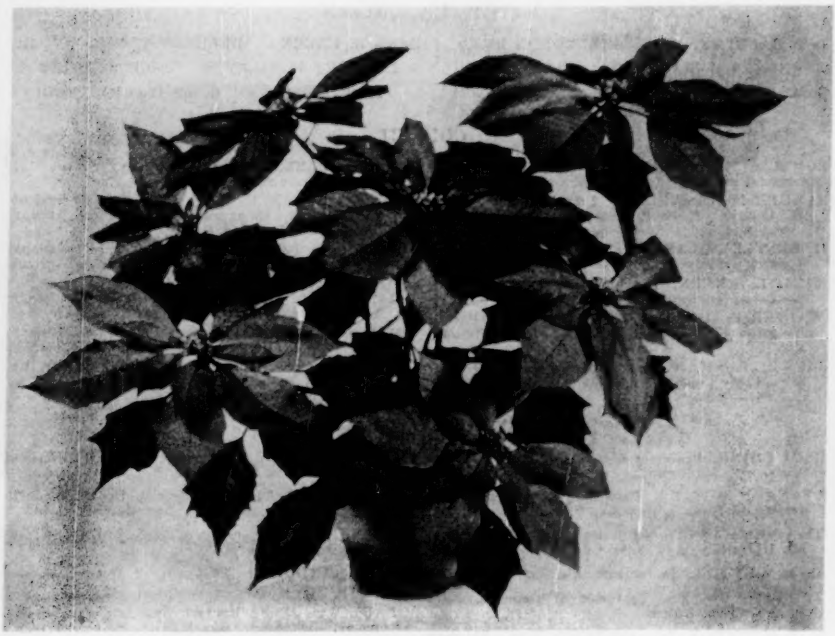
The Journal of HEREDITY



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Wild Barley Hybrids

Factors in "Taste-Blindness"

Second List of Tomato Mutants

The "Splitfoot" Lethal in the Fowl

VOLUME 51 JULY-AUGUST, 1960 NUMBER 4

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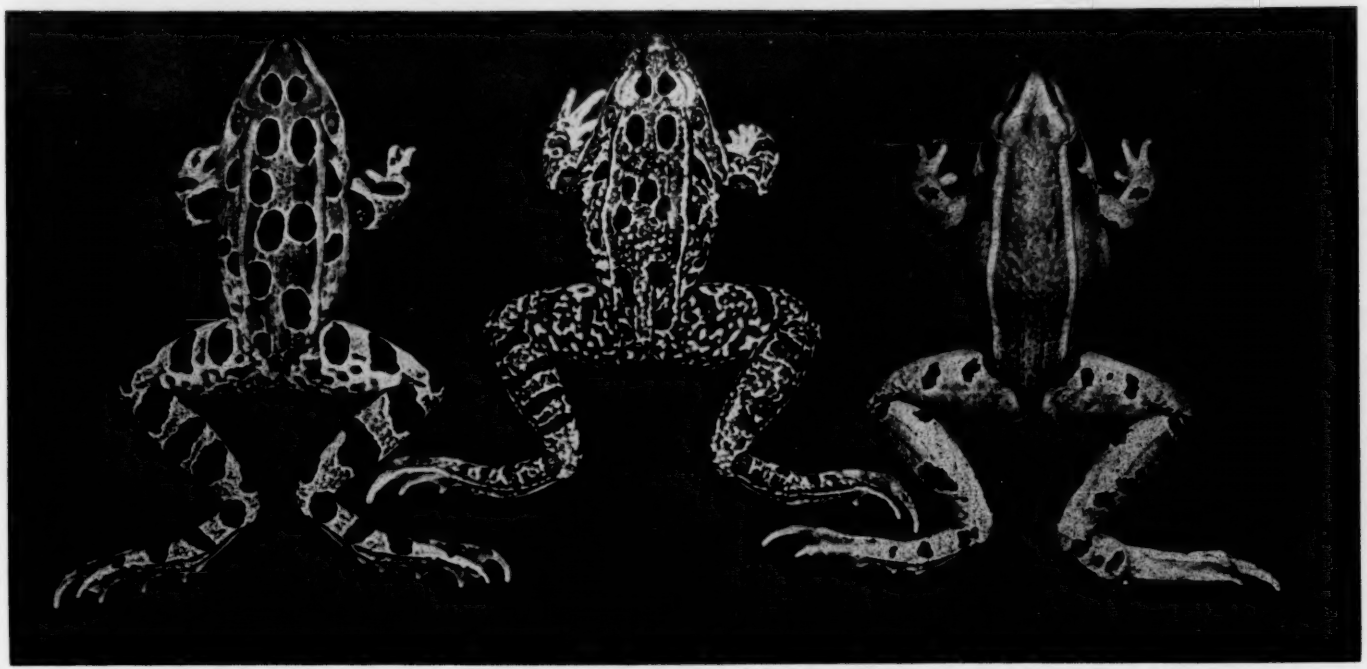


VARIANT PATTERNS OF LEOPARD FROGS

Figure 1

Two variant patterns, kandiyohi or mottled (*B*) and burnsi or nonspotted (*C*), are simple dominants to the wild-type spotted or *pipiens* pattern (*A*).

INTERACTION OF MUTANT GENES IN THE LEOPARD FROG

E. PETER VOLPE*

TWO mutant genes have been disclosed that affect the dorsal pigmentary pattern of the common leopard frog, *Rana pipiens* (Figure 1). The nonspotted or burnsi gene results in the absence of dorsal spots that typify the common leopard frog[3, 9]. The mottled or kandiyohi gene is responsible for vermiculate mottlings in the interspaces between the dorsal spots[7, 9]. The variant genes are each dominant to the wild-type spotted or *pipiens* allele but lack dominance with respect to each other[1, 8]. The double dominant frog, designated phenotypically as "mottled burnsi," combines the characteristics of the two mutant forms.

Mottled burnsi offspring have been derived from the experimental cross of a heterozygous kandiyohi and a heterozygous burnsi frog[1]. The offspring of this cross segregate as follows: 1 *pipiens* : 1 burnsi : 1 kandiyohi : 1 mottled burnsi. The segregating ratio does not permit a distinction between multiple allelism (or pseudoallelism†), linkage, or independent assortment. Assuming a multiple allelic series (burnsi or nonspotted, C^B; kandiyohi or mottled, C^K; *pipiens* or spotted, c^+), the heterozygous kandiyohi and burnsi parents would be C^Kc^+ and C^Bc^+, respectively. The mottled burnsi offspring would represent the compound, C^KC^B. The 1:1:1:1 ratio can be explained equally well by assuming two pairs of alleles that assort independently or are linked. Designating the kandiyohi gene, *K*, burnsi, *B*, and *pipiens*, K^+ or B^+, the kandiyohi parent could have been either $KK^+B^+B^+$ (independent assortment) or KB^+/K^+B^+ (linkage). Similarly, the burnsi parent could have been either $K^+K^+BB^+$ or K^+B/K^+B^+. The mottled burnsi offspring would be either KK^+BB^+ or KB^+/K^+B.

A clue to the mode of inheritance of the varieties resides with the double dominant frog. The types and ratio of offspring resulting from a testcross of the mottled burnsi and the recessive wild-type *pipiens* frog would serve to differentiate between the alternatives. Attempts to rear the mottled burnsi offspring to sexual maturity were not successful. More disconcerting was the apparent absence of mottled burnsi frogs in natural populations. There were no published reports of the existence of such a frog in nature.

Knowledge of the natural distribution and frequencies of the kandiyohi and burnsi mutants is incomplete[2, 5, 9, 10]. Localities from which the kandiyohi variant has been recorded are the southwestern portion of Minnesota and the eastern border of South Dakota. The center of distribution of the burnsi mutant is also southern Minnesota, with peripheral populations in the adjacent states of South Dakota, Iowa, and Wisconsin. All three forms (kandiyohi, burnsi, and the wild-type, *pipiens*) are known to occur together in certain localities. Professor David J. Merrell (University of Minnesota) informed me that he and Professor James C. Underhill (University of South Dakota) captured a burnsi and a kandiyohi frog within approximately 200 feet of each other at Clear Lake, Marshall County, South Dakota. In another personal communi-

*Department of Zoology, Newcomb College of Tulane University, New Orleans, Louisiana. This investigation was supported by a grant from the National Science Foundation (NSF-G7080).

†Closely linked genes, rarely fractionable by crossing-over, will, of course, simulate a multiple allelic series.

PROGENY FROM KANDIYOHI × BURNSI CROSS

Figure 2

The double dominant frog, designated phenotypically as "mottled burnsi" (*B*), combines the characteristics of the two dominant mutants, kandiyohi or mottled (*A*) and burnsi or nonspotted (*C*). Frog *D* possesses a highly modified form of the burnsi pattern.

cation, Professor J. Frank Cassel (North Dakota Agricultural College) stated that he observed the three forms (kandiyohi, burnsi, and *pipiens*) at Bad Axe Lake, Hubbard County, Minnesota. Hence, a chance meeting of a kandiyohi and a burnsi frog should produce the double dominant frog. Unless the mottled burnsi frogs are at a very strong selective disadvantage in nature, they should be present in populations in which the kandiyohi and burnsi variants coexist. It recently became evident that the naturally occurring mottled burnsi frog had simply escaped detection.

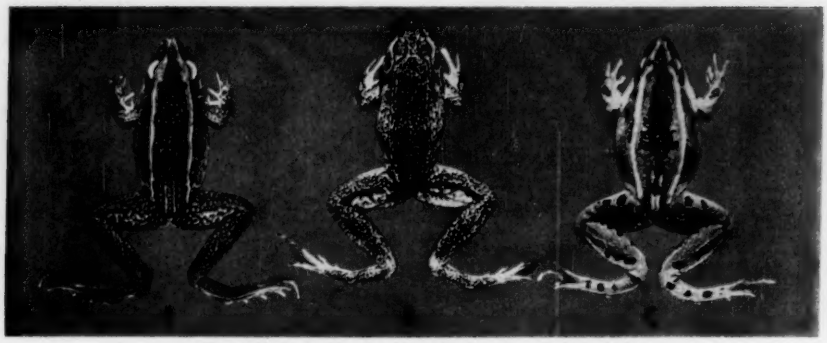
In the fall of 1958, Mr. Steinhilber (professional dealer, Steinhilber Frog Farm, Oshkosh, Wisconsin), after a diligent search at my request, uncovered two adult frogs in the eastern part of South Dakota that resembled the mottled burnsi frogs recovered from laboratory crosses. A third frog, rather distinctive in appearance, was also shipped to my laboratory. The three frogs were testcrossed to the recessive *pipiens*, the results of which are presented herein.

Recognition of Variant Frogs

The phenotypic expressions of the mottled or kandiyohi gene are more or less uniform. As seen in the adult (Figure 1*B*), the inter-spot pigment blotches pervade the entire dorsal surface, including the appendages. Young kandiyohi offspring (Figure 2*A*) from laboratory crosses, reared beyond metamorphosis, are readily identifiable.

The nonspotted or burnsi gene has variable expressivity. Burnsi frogs may be devoid of dorsal black spots or contain a few to several spots, particularly on the legs, varying in size and shape. A comparison of burnsi frogs shown in Figures 1*C* and 2*C* permits some notion of the extent of variation. Both frogs illustrated are heterozygous (BB^+). The adult burnsi with spotted appendages had been tested in an earlier cross[8]; the young burnsi, virtually unmarked, resulted from the cross of a heterozygous kandiyohi (KK^+) and a heterozygous burnsi (BB^+), previously reported[1]. The latter cross, as mentioned in the introduction, yielded the mottled burnsi frog. The features of the young mottled burnsi are contrasted with those of the kandiyohi and burnsi offspring in Figure 2. The mottled burnsi frog lacks the large dorsal spots, as does the unmarked burnsi mutant, and possesses the vermiculate mottling of the kandiyohi deviant.

The offspring represented in Figure 2*D* was also derived from the aforementioned cross (heterozygous kandiyohi × heterozygous burnsi). This offspring was classified as a burnsi, albeit a highly modified burnsi. The view was



VARIANT PATTERNS UNCOVERED IN NATURE

Figure 3

Breeding experiments have revealed that *A* and *B* are double dominant or "mottled burnsi" frogs; *C* is a burnsi frog, albeit a highly modified burnsi.

taken that the highly modified pattern was due to either variability in the penetrance of the burnsi gene or the action of genetic modifiers. However, in the absence of further breeding tests, this so-classified highly modified burnsi offspring could conceivably have been a mottled burnsi frog. The melanophore patches on the dorsum resemble, perhaps superficially, the pigment blotches of the mottled burnsi frog. The results of the present investigation, discussed below, support the contention that the frog represented in Figure 2*D* is genetically a burnsi, and not a mottled burnsi.

Breeding Experiments

The three adult frogs received from Mr. Steinhilber are shown in Figure 3. Two, a female (3*A*) and a male (3*B*), were considered as mottled burnsi frogs prior to experimentation. The third, a female (3*C*), was thought to be a highly modified burnsi frog, comparable to the aforementioned offspring encountered in laboratory crosses (*cf.*, Figures 2*D* and 3*C*). However, the possibility existed that the dorsal pattern was rather a modified form of the mottled burnsi pattern.

Eggs of female "3*A*" were inseminated by sperm of a wild-type *pipiens* male. Reciprocally, the eggs of a wild-type *pipiens* female were fertilized by sperm of the male frog "3*B*." Female frog "3*C*" was also testcrossed to a wild-type *pipiens*. Fertilized eggs were obtained by the routine procedure[4] of

TABLE I. Results of testcrosses with the common leopard frog, *Rana pipiens*

Matings	Phenotypes of offspring: *pipiens* Obs.	*pipiens* Exp.	burnsi Obs.	burnsi Exp.	kandiyohi Obs.	kandiyohi Exp.	mottled burnsi Obs.	mottled burnsi Exp.	χ^2	P
A. Frog "3*A*" ♀										
× *pipiens* ♂	20	19.00*	24	19.00	18	19.00	14	19.00	2.74	>30%
		17.48†		20.52		20.52		17.48	1.96	>50%
B. *pipiens* ♀										
× frog "3*B*" ♂	25	20.50*	22	20.50	19	20.50	16	20.50	2.20	>50%
		18.86†		22.14		22.14		18.86	2.88	>30%
C. Frog "3*C*" ♀										
× *pipiens* ♂	40	38.00	36	38.00	—		—		0.21	>50%

*Expected on the basis of two pairs of independently assorting genes.
†Expected on the basis of loose linkage (postulated 46 percent cross-over value).

inducing ovulation by pituitary injection and subsequently "stripping" the eggs of the gravid female into a sperm suspension. The latter was prepared by crushing the pair of testes in 20 cc. of 0.10 Ringer's solution.

Embryos from each cross were distributed in groups of eight in white enamel pans (12″ × 8″ × 2″) containing pond water. Commencing with the larval period (stage I, defined and illustrated in Taylor and Kollros[6]), the tadpoles were fed pieces of boiled spinach. The pond water was changed at two- to three-day intervals; a fresh supply of spinach was provided at each change period. When the tadpoles approached metamorphosis, they were transferred to terraria containing pond water at one end. The juvenile frogs were fed vestigial-winged fruit flies for a period of at least one month after transformation, then classified and preserved in 0.10 formalin for future reference.

The results of the experiments are summarized in Table I. The "3*A*" ♀ × *pipiens* ♂ cross yielded 76 metamorphosed young, of which 26.4 percent were *pipiens;* 31.6 percent burnsi; 23.6 percent kandiyohi; and 18.4 percent mottled burnsi. Four patterns were also found among the 82 offspring of the reciprocal cross, *pipiens* ♀ × "3*B*" ♂, the percentage of each phenotype being as follows: 30.5 percent *pipiens,* 26.8 percent burnsi, 23.2 percent kandiyohi, and 19.5 percent mottled burnsi. Neither of the two segregating ratios differs significantly from 1:1:1:1. As might have been expected, the most variable pattern was that of the burnsi offspring, although separable from those of the others. The results are interpretable on the basis that the test frogs, "3*A*" and "3*B*," were indeed double dominant or mottled burnsi frogs, each heterozygous for the two pairs of mutant genes.

The metamorphosed young from the "3*C*" ♀ × *pipiens* ♂ cross showed a sharply defined 1:1 segregation into the *pipiens* pattern and the burnsi pattern. Consequently, the test female "3*C*," although containing melanophore patches on the dorsum, was a heterozygous burnsi frog (BB^+). Several of the burnsi offspring resembled the burnsi parent.*

Interpretations

The double dominant frogs (mottled burnsi) tested were each heterozygous for both pairs of genes (KK^+BB^+). The results of testcrosses of mottled burnsi frogs to the recessive wild-type *pipiens* ($K^+K^+B^+B^+$) suggest that the two mutant genes, kandiyohi (K) and burnsi (B), are located at two different, independently assorting loci. Although the possibility of multiple alleles is effectively dismissed by the present data, the interpretation of linked genes still lingers. If the burnsi and kandiyohi genes in the heterozygous mottled burnsi frogs tested are loosely linked, i.e., located far apart on the chromosome, then independent assortment is simulated. Postulating a cross-over value of 46 percent, and assuming the repulsion phase, the cross of a mottled burnsi (KB^+/K^+B) and a *pipiens* (K^+B^+/K^+B^+) would yield the following: 0.23 *pipiens* : 0.27 burnsi : 0.27 kandiyohi : 0.23 mottled burnsi. The segregation actually observed in the progeny of such a cross (Table I) does not differ significantly from this hypothetical linkage ratio. Nevertheless, it should be noted that there is a slight deficiency of only one crossover class, the mottled burnsi class. There is no corresponding deficiency of the *pipiens* recombination class. However, the numbers of progeny recovered in the crosses are too few to assert that the *pipiens* recombinants are truly in excess, if linkage in the repulsion phase is involved.

Although the possibility of loose linkage cannot be discounted, the interpretation favored is that of independent assortment. The slight deficiency of mottled burnsi frogs may reflect some selective disadvantage in laboratory cultures. This is speculative, as the breeding experiments were not designed to measure differential viability.

Summary

Two pattern variants, kandiyohi and burnsi, are simple dominants to the common leopard frog, *Rana pipiens,* but lack dominance with respect to each other. Hitherto, the double dominant frog, designated mottled burnsi, had been recovered only from laboratory cultures; it apparently had eluded naturalists in the past. The query as to whether the kandiyohi and burnsi genes arose as mutations at the same

*Heavily spotted burnsi frogs are being investigated further. Preliminary results suggest that modifiers (genes with small effects) determine the magnitude of expression or dominance of the burnsi gene.

locus or at different loci would be answered by appropriate matings utilizing the mottled burnsi frog. The recent uncovering of two mottled burnsi adult frogs in nature and subsequent testcrossings to wild-type, recessive *pipiens* frogs revealed that the mutant genes are not allelic. The kandiyohi and burnsi genes are located at different loci, but independent assortment of the mutant loci has not been unequivocably demonstrated. Widely separated loci on the same chromosome (e.g., hypothetical linkage of 46 percent) would simulate independent assortment.

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INSEMINATION pH AND THE SEX RATIO IN RABBITS

C. W. EMMENS*

VARIOUS claims have been made that by douching the vagina of domestic or laboratory animals with lactic acid or sodium bicarbonate before mating, the sex ratio may be modified. Such claims state that acid conditions produce an excess of females and alkaline conditions an excess of males. During the period from 1930 to 1942, a series of contradictory papers appeared from which a general impression is gained that there may be little or no truth in the belief that vaginal pH affects the sex ratio. The position is summarized by Weir[3], who concluded that, although the possible relation of vaginal pH to the sex ratio is wholly speculative, there was a connection between blood pH and sex ratio in two related lines of mice. These lines were selected for blood pH, the high line (pH 7.466) giving significantly more males (percent) than the low line (pH 7.420). This is in the direction expected from such previous tests as had reported positive results.

It is not intended to review the literature on the "acid-alkali" problem here. All reported tests but those of Weir[3] and Cole *et al.*[1] have been of pH changes in the vagina, or the blood, or of trials with acid or alkali in the diet. Cole *et al.*[1] made a few tests in which the semen of rabbits was treated with lactic acid or sodium bicarbonate prior to artificial insemination. They obtained no offspring at pH 3.8 to 5.1, 29 males and 35 females at pH 5.6 to 6.2, and 20 males and 23 females at pH 7.9 to 9.2. The altered semen was held at room temperature for 15 minutes, occasionally one hour, before insemination.

The present tests were made during 1946 and 1947 while the author was at the National Institute for Medical Research, London, but were not then reported because they were completely negative. However, since they are quite extensive and have not been duplicated elsewhere, and since some interest in the subject is again being shown, it was decided to publish them.

Materials and Methods

Female Dutch-marked or Himalayan rabbits were injected intravenously with 20 I.U. of chorionic gonadotrophin at about 9:30 a.m. Semen from males of the same breeds was collected, pooled, split and diluted with isotonic buffered solutions at about 3 p.m., usually at a 1:10 dilution. Acid buffers were 0.1 *M* sodium phosphates in sodium chloride and dextrose at pH 5.2 or 6.0; alkaline buffers were 0.1 *M* sodium carbonate-bicarbonate in sodium chloride and dextrose at pH 9.8. The buffer-semen mixtures varied in actual pH from 5.4 to 6.2 or from 9.0 to 9.8, with nearly all alkaline samples over 9.5.

The diluted semen was then left to stand either at room temperature or, in a few instances, at 38° C. for from one to three hours, most often for 1½ to 2½ hours, and its motility was observed at the beginning and end of this period. The pH changed little or not at all during this period. A motility score[2] was assigned according to a subjective system of assessment in which 4 represents maximum motility and 0 immotility. Initial motility was nearly always good, score 3 or better, and only preinsemination motilities are described below.

Equal numbers of does were inseminated with acid and alkaline diluted semen at 5 to 6 p.m. The insemination volume was 2 ml. per female. The young were sexed at birth and the females reused after an interval. Untreated control semen was not used since a contrast of the effects of the two treatments was the point at issue.

*Department of Veterinary Physiology, The University of Sydney, N. S. W., Australia.

Results

A total of 262 inseminations was performed with 140 females, which resulted in the birth of 156 litters and 620 young. The results are shown in Table I, grouped according to the final motility of the semen. It was felt that this might affect fertility, and possibly the sex ratio. Since no over-all effects were seen, the temperature at which the semen was held is ignored in this table and on separate examination was seen to have had no signiflcant effect. Calculation of $\chi^2 = 6.81$, d.f. $= 11$, $P = 0.80$ for the proportion of males in each row of the table shows that the results of insemination grouped according to pH and the motility of the semen prior to insemination are homogeneous and that no significant treatment effects occurred. The value $\chi^2 = 15.92$, d.f. $= 11$, $P > 0.10$ also shows that although the percentage of rabbits pregnant dropped with lowered motility in acid conditions, this cannot be considered significant on the present figures. The finding is, however, in line with the expectation that at some point the spermatozoa must be damaged or killed by extreme or prolonged pH changes or both. It is also in agreement with observations[2] that rabbit spermatozoa may be revived after becoming immotile in acid buffers. No completely immotile alkaline suspensions were seen. It is of interest that the size of litters was unaffected by the treatments over the range investigated.

Discussion

These results are in complete agreement with the less extensive tests of Cole *et al.*[1], and were obtained with somewhat greater extremes of pH and after longer exposure times. It is difficult to suppose that pH changes in the female genital tract can affect the sex ratio of the offspring when prolonged exposure of spermatozoa to physiological extremes of pH has such negative effects. It remains possible, however, that due to differences in iso-electric points, differentiation between X- and Y-bearing sperm could occur at some intermediate pH. The various grades of motility may be a rough measure of the extent to which the pH treatment affected the pooled semen samples. However, this did not involve factors significantly influencing fertility or the sex ratio over the range investigated.

Summary

Exposure of diluted rabbit semen to a pH of 5.4 to 6.2 or of 9.0 to 9.8 for one to three hours prior to insemination did not affect the sex ratio of the offspring.

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TABLE I. Independence of pregnancy rate, litter size and sex ratio of the pH and terminal motility of diluted rabbit semen

Motility	No. ♀	No. pregnant	% pregnant	Offspring ♂	Offspring ♀	Mean litter size
pH 5.4-6.2						
0	6	2	33	3	6	4.5
½	19	8	42	16	15	3.9
1	12	5	42	7	14	4.3
1½	39	27	69	55	55	4.1
2	24	17	71	31	36	3.9
2½	19	11	58	19	16	3.2
3	12	7	58	13	13	3.7
Total:	131	77	59	144	155	3.9
pH 9.0-9.8						
½-1	7	4	57	7	5	3.0
1½	21	12	57	25	25	4.2
2	38	27	71	59	50	4.0
2½	45	25	56	45	59	4.2
3	20	12	60	24	22	3.8
Total:	131	79	60	160	161	4.1
Grand Total:	262	156	60	304	316	4.0

VIBRATOR, A RECESSIVE SEX-LINKED MUTATION IN TURKEYS

T. H. Coleman, R. K. Ringer, W. J. Mathey, K. G. Rood and C. W. Pope*

DURING the 1956 hatching season, three Broad Breasted Bronze poults hatched at this institution exhibited a very rapid shaking, or vibration, of the head and neck. In the first hatch of 1957, there were sufficient numbers of affected poults to suggest that a pathogen may have been responsible, and appropriate laboratory procedures were followed in an unsuccessful attempt to isolate the causative organism. When the affected poults were sexed by a commercial sexer, more than 95 percent were females.

In subsequent hatches all "vibrators" were from eggs produced by females in three single-male mating pens. The three males heading these pens were full brothers. Approximately one-half of the female progeny of these three sires were vibrators, but, when examined, the sires did not exhibit the vibrating condition. However, the dam of these sires was still available and was found to be a vibrator. The two dams which produced vibrating male progeny during the 1957 hatching season were also found to be vibrators.

Materials and Methods

When the above information was considered, the presence of a sex-linked recessive gene was indicated. To test this theory, vibrators and normal turkeys were mated in all possible combinations during the 1958 hatching season. Sex was determined either by sacrificing day-old poults for internal examination or by observing external secondary sexual characteristics after maturity.

Results

All progeny produced by mating a normal male to normal females were, as expected, normal (Table I). The sex ratio of the progeny (13 males : 11 females) approached expectancy.

The mating of a normal male to vibrator females produced 78 poults; all were normal. The sex ratio of those progeny which were sexed was 25 males : 24 females.

Twenty progeny were produced from the mating of a vibrator male to normal females. Eleven of the 20 poults were normal; nine were vibrators. All normal progeny were sexed and found to be males. All sexed vibrators (six) were females. The above ratios conform to those expected when a single sex-linked recessive gene is responsible for a character.

Thirty-nine progeny, all vibrators, were produced from the mating of a vibrator male to vibrator females. The sex ratio of those sexed was 17 males : 13 females.

When a heterozygous vibrator male was mated to normal females, 40 offspring were obtained. Thirty of these offspring were normal and 10 (exactly 3:1) were vibrators. The ratio of those normal progeny which were sexed was 14 males : 12 females. Nine vibrators were sexed; all were females. Again, a study of the evidence indicates that a sex-linked recessive gene is responsible for the vibrator condition.

The mating of a heterozygous male to vibrator females produced 60 progeny, 29 being normal and 31 vibrators. The ratio of those normal progeny which were sexed was 8 males : 7 females, and that of the vibrator progeny was

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11 males : 10 females. Once more the results are indicative of a sex-linked recessive gene.

Results of the 1958 matings prove rather conclusively that the vibrator condition is caused by a single sex-linked recessive gene. The symbol *vi* is suggested for this character.

Discussion

Hereditary nervous conditions have been reported in various animals, including humans, mice, rats and cattle. Several hereditary nervous conditions have been reported in chickens. The first of these was described by Knowlton[5]. He reported that affected chicks hatched normally but could not stand more than a few seconds, and died within a few days. The condition was found to be inherited as an autosomal recessive. Knowlton named the condition "congenital loco."

Hutt and Child[4] described a condition in chickens which they called "congenital tremor." At hatching time, affected chicks showed varying degrees of tremor, and over two-thirds of the tremblers died within one week. Only a very small percentage of them lived beyond six months of age. Hutt and Child found the condition to be hereditary; however, the genetic basis was apparently complex.

A condition was reported by Bohren[1] and by Godfrey *et al.*[3] in which affected chicks exhibited a retraction of the head over the back accompanied by a rapid shaking of the head. The condition was highly lethal, with only about one percent of the affected females reaching maturity. This disorder, which was named "jittery," was inherited as a sex-linked recessive.

Scott *et al.*[6] reported a disorder in chickens which they called "shaker," in which affected birds showed rapid movements of the head and neck with progressively increasing difficulty in walking without stumbling. The disorder was not discernible in newly hatched chicks; however, it was manifested as early as 18 days of age. Only a few affected females lived to sexual maturity. This condition was inherited as a sex-linked recessive.

Before the appearance of "vibrator," only one hereditary nervous condition had been reported in turkeys. Cole[2] described this condition caused by an autosomal recessive gene and identical in all known respects with the one initially described in chickens by Knowlton[5]. Affected poults hatched in excellent physical condition, but lived no longer than a week even when fed and watered by hand. Because of its similarity to the condition reported by Knowlton, Cole called the condition in turkeys by the same name, "congenital loco."

Vibrator differs markedly from any hereditary nervous condition previously reported in chickens or turkeys in that it is not lethal. Survival of growing and adult birds is apparently equal to that of their normal counterparts. One- and two-year-old birds appear normal in all respects other than a vibration, which can be detected by holding the head and/or neck of the turkey loosely in the hand. From the standpoints of egg production, fertility and ability to hatch, reproductive ability is not appreciably decreased in vibrator females when compared to normal females. The effect of the condition on male reproductive ability has not been determined.

The oldest vibrators were discovered in nonpedigreed foundation stock secured from a local turkey hatcheryman in 1955. Their vibrating was not observed until they were examined as adults during the 1957 hatching season. As the local hatcheryman secured breeding stock each year from one of the country's larger Broad Breasted Bronze breeders, it was impossible to determine where the mutation occurred.

Summary

A very rapid shaking, or vibration, of the head and neck was noted in Broad Breasted Bronze poults hatched in 1956. Analysis of 1957 hatching records pointed to the action of a single sex-linked recessive gene as being responsible for the condition. Vibrators and normal turkeys were mated in all possible combinations during the 1958 hatching season. Results obtained from these matings proved

TABLE I. Results of mating normal and vibrator turkeys in all combinations

Mating	Progeny					
	Normals			Vibrators		
	♂♂	♀♀	N. S.*	♂♂	♀♀	N. S.*
Normal ♂ × normal ♀♀	13	11				
Normal ♂ × vibrator ♀♀	25	24	29			
Vibrator ♂ × normal ♀♀	11				6	3
Vibrator ♂ × vibrator ♀♀				17	13	9
Heterozygous ♂ × normal ♀♀	14	12	4		9	1
Heterozygous ♂ × vibrator ♀♀	8	7	14	11	10	10

*Not sexed

the earlier theory. The symbol *vi* is suggested for the character.

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LALOR FOUNDATION RESEARCH AWARDS

The Lalor Foundation has announced the program of awards for 1961 which it is offering for support of research on the fundamental biochemical and physiological mechanisms concerned with fertility and the early stages of reproduction in various forms of life. The objectives are to further the knowledge and understanding of the basic phenomena involved and to extend and develop the possibilities for effective regulation and control.

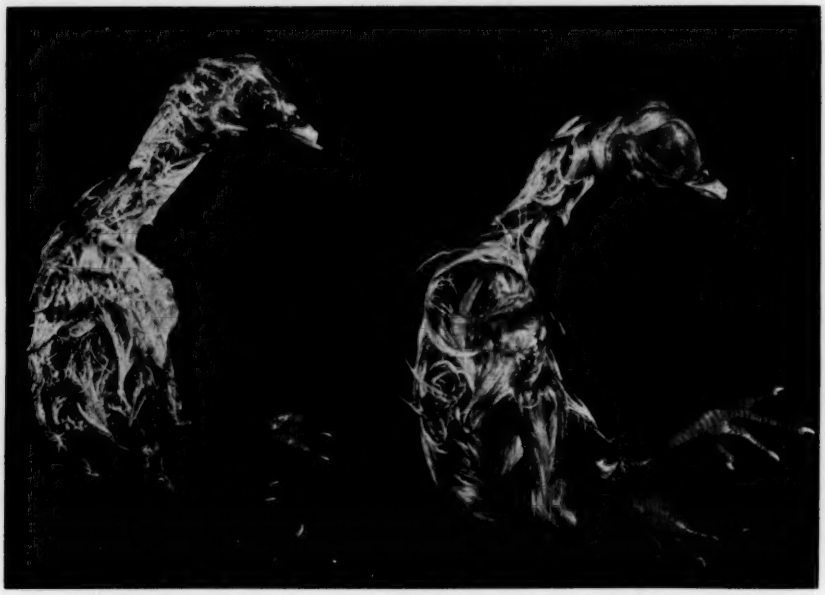
The awards may range up to $8,000 per year depending upon the scope and duration of the projects approved. Preference will be given to younger members of university and college faculty and staff with an upper age limit of 41 years. The work may be carried on at the applicant's own institution or elsewhere.

The Foundation will also grant post-doctorate summer or short-term research awards at the Marine Biological Laboratory at Woods Hole, Massachusetts, or elsewhere for appropriate projects in the fields specified. These awards will normally not exceed $1,000 for a single man or a woman, $1,200 for a married man working at his home institution, and $1,350 for a married man with principal program at another institution.

Requests for information and for application forms should be directed to the Lalor Foundation, 4400 Lancaster Pike, Wilmington 5, Delaware. The final date for receipt of executed application forms complete with supporting data is January 16, 1961. Notification of appointment will be on or before March 15.

SPLITFOOT, A LETHAL MUTATION AFFECTING THE FOOT STRUCTURE OF THE FOWL

U. K. Abbott and P. F. Goetinck*



ABNORMALITIES IN A SPLITFOOT EMBRYO

Figure 4

Splitfoot (left) and normal (right) full-sib embryos sacrificed on the eighteenth day of incubation. Note feet, reduced pre-maxilla, abnormal eye and slight micromelia. ×1.

FOUR embryos which appeared to have two pairs of identical digits per foot were found in March 1958, during the course of routine examination of unhatched embryos from an inbred line of White Leghorn chickens. Two full brothers mated to their full sisters produced these deviant types; one was found among 12 identified progeny of male 236 × female 231 and three among 11 progeny of male 228 × female 223.

This phenotype had not appeared previously in any line maintained at this station. Test matings made to establish the possible mode of inheritance of the abnormality revealed considerable variability in its phenotypic expression. A foot had from three to five digits; these were sometimes accompanied by additional toenails and tarso-metatarsal bones. Beak structure and eye development were also affected. In a few in-

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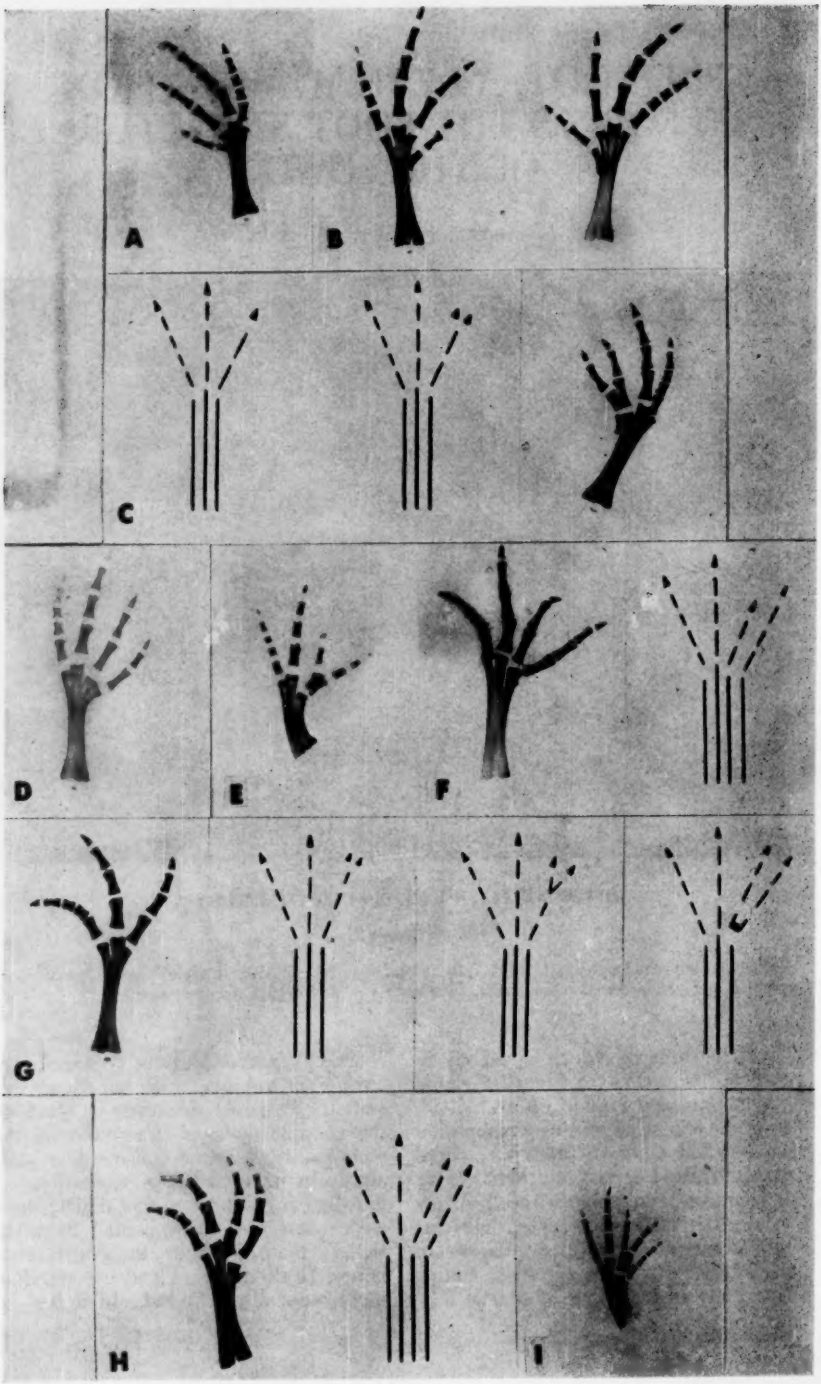
A
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C
D
E
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G
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I

stances, small cyst-like structures were scattered over the epidermis of the thigh and wing. The new phenotype was termed splitfoot, to describe the doubling of two normal digits observed in the first cases.

Several hereditary variations in the foot structure of the domestic fowl are discussed by Hutt[2]. These include the dominant gene *Po* for polydactyly, its allele duplicate, and the autosomal recessive genes for diplopodia, talpid and talpid^2.[1] The most common expression of polydactyly involves the addition of one digit (closely resembling the hallux) to the pre-axial side of the foot. Polyphalangism is another expression of polydactyly, in which the normal number of toes are present but the hallux possesses one additional phalanx. In duplicate the first digit may be split in two or be replaced by three toes. Frequently the tarso-metatarsus may be doubled as well. In the most common expression of diplopodia, the hallux is replaced by three toes articulating with three extra tarso-metatarsal bones. In both talpid mutants the entire foot structure is altered; embryos may have as many as 10 syndactylous digits per foot. Among other characteristics, these three mutants have in common supernumerary wing fingers, a reduced pre-maxilla and a smaller body size.

Morphology and Classification

The foot of a normal chicken has four digits. The toes are numbered mediolaterally. The first toe, or hallux, has two phalanges; digits II, III and IV have three, four and five phalanges, respectively (Figure 5). Splitfoot embryos may have three, four or five toes, which may or may not include the hallux. The digits differ in their orientation and in numbers of phalanges and tarso-metatarsal bones present. The two feet may differ in digit number. Among 136 feet examined as alizarin preparations, 18 different patterns of foot expression were found. An arbitrary grouping of the morphologically different feet was adopted in an attempt to analyze the phenotypic variation observed and to investigate the embryological action of the gene. Among the specimens studied as alizarin preparations, the third and fourth digits were invariably normal in both orientation and number of phalanges. In some cases both digits I and II were affected, while in others the phenotypic effect was limited to the hallux. Effects on the foot could be classified in five main groups as follows: 1) only the hallux was affected; 2) the second toe apparently was split in two, each digit having three phalanges; 3) the second toe was split, but the lateral bifurcation had three phalanges while the median one had four; 4) both parts of the second toe had four phalanges; and 5) the foot possessed five digits (two digits were supernumerary since the hallux was absent).

Saunders[3] has shown that without the presence of the apical ectoderm there

NORMAL AND SPLITFOOT EMBRYONIC FEET

Figure 5

All specimens are between 17 and 19 days of incubation. Diagrams represent patterns observed but not photographed. ×1.05. *A*—Normal. *B*—Only the hallux is affected through either its duplication or elongation; in the latter case the phenotype resembles polyphalangism. *C*—The hallux may be completely absent, giving a three-toed foot. The normal second toe may be incompletely duplicated with a basic number of three phalanges in both sections. *D*—Duplication of the second digit is complete, resulting in two second toes, each possessing three phalanges. Rudimentary parts of the hallux may be present. *E*—The second toe is split distally, having a basic number of three phalanges in the lateral and four in the median bifurcation. *F*—Bifurcation of the digit is complete and may extend into a duplication of the second tarso-metatarsal bone as well. *G*—The hallux may be completely absent, resulting in a three-toed foot, while the second digit possesses four phalanges. This digit may be split distally. *H*—Splitting of digit II is complete, resulting in two second toes with four phalanges each. The second tarso-metatarsal bone may be duplicated. *I*—The foot lacks the hallux but has five digits. The second digit is present in triplicate. The second tarso-metatarsal bone is duplicated as well.

can be no distal development of the embryonic limb bud of the chick. Zwilling[4] has emphasized the interaction and reciprocal relationship between the mesoderm and ectoderm, in that the latter is maintained in an active (functional) condition by a mesodermal factor. Zwilling and Hansborough[5] have suggested that polydactylous mutations may affect the mesodermal factor, resulting in an increase in the area of active pre-axial ectoderm.

If the distribution of an ectoderm maintenance factor is involved in the abnormal digit differentiation encountered in the present case, either variation in time of function or a change in its spatial distribution might be considered as an explanation of gradations in expression. Since the hallux is the last

TABLE I. Incidence of associated abnormalities in splitfoot and normal progeny

a) Comparison of original and test matings

	Splitfoot					Normal				
	Original (41)		Test (47)			Original (117)		Test (86)		
Abnormality	No.	%	No.	%	χ^2c	No.	%	No.	%	χ^2c
Reduced pre-maxilla	30	73.2	34	72.3	0.02	0	0.0	0	0.0	0.00
Incomplete eyelid	27	65.8	39	82.9	2.57	0	0.0	0	0.0	0.00
Residual albumen	10	24.4	16	34.1	0.57	2	1.7	1	1.2	0.07
Malposition	13	31.7	4	8.5	2.36	13	11.1	3	3.5	0.015
Micromelia	12	29.3	2	4.3	8.46*	5	4.3	2	2.3	0.13

b) Combined matings

	Splitfoot (88)		Normal (203)		
Abnormality	No.	%	No.	%	χ^2c
Reduced pre-maxilla	64	72.7	0	0.0	185.06*
Incomplete eyelid	66	75.0	0	0.0	192.67*
Residual albumen	26	29.5	3	1.5	50.83*
Malposition	17	19.3	16	7.8	14.59*

*P < .01

TABLE II. Proportions of normal, splitfoot and unidentified (early dead) progeny of original matings and test matings

					Progeny phenotypes				
					Normal	Splitfoot			
Mating	Dam	Number eggs set	Fertility No.	Fertility %	Number unidentified	No.	No.	% of identified	χ^2c 3 normal: 1 splitfoot
Original	0-12	66	40	60.6	6	29	5	14.7	1.41
	0-13	76	56	73.7	8	34	14	29.2	0.25
	0-14	81	57	70.4	17	27	13	32.5	0.83
	0-16	59	40	67.8	4	27	9	25.0	0.00
Subtotal	1	282	193	68.4	35	117	41	25.9	0.03
Test	0-12	44	35	79.5	4	20	11	35.5	1.30
	0-13	34	19	55.9	4	10	5	33.3	0.20
	0-14	50	17	34.0	6	9	2	18.2	0.03
	0-16	25	14	56.0	1	11	2	15.4	0.23
	2171	29	28	96.5	16	6	6	50.0	2.77
	2169	29	20	68.9	14	2	4	66.7	3.55
	2170	35	33	94.3	7	13	13	50.0	7.38†
	1360	22	22	100.0	3	15	4	21.1	0.02
Subtotal	2	268	188	70.1	55	86	47	35.3	7.04†
Grand total		550	381	69.3	90	203	88	30.2	3.99*

*P < .05

†P < .01

toe to differentiate in normal embryos, the occurrence of phenotypes with only the first toe affected might suggest a later time of onset of activity, while the more severe expressions leading to a transgression of the abnormality from the first to the second digit could be explained by earlier action of this factor. A classification based on these assumptions is presented in Figure 5.

Age at Death

A score of 21 days was given to living embryos which had not pipped the shell by the twenty-second day when the hatch was removed from the incubator. Embryos which had pipped the shell but had not hatched received a score of 22. Only four among 88 splitfoot embryos, from both the original and backcross matings, fell in the latter category. The mean time of death in the original matings was 18.8±.4 days. In the test matings it was 17.3±.5 days.

Associated Abnormalities

Other abnormalities found to be characteristic of splitfoot embryos include: 1) reduced pre-maxilla; 2) an incomplete eyelid, at times accompanied by blindness and cataract; 3) substantial amounts of residual albumen, which in normally developing embryos is completely utilized by the seventeenth day of incubation; and 4) micromelia (Figure 4 and Table I).

Splitfoot embryos from the original matings had a higher incidence of micromelia than their normal siblings ($\chi^2c = 17.23$, $p < .01$). However, this difference disappeared in the comparison between splitfoot and normal siblings from the test matings ($\chi^2c = .009$). The difference between the incidence of micromelia in splitfoot embryos from the original and the test matings was also highly significant ($\chi^2c = 8.46$, $p < .01$).

Two hens produced a few splitfoot embryos but no normal embryos with small cyst-like structures scattered over the epidermis of the wings and thighs. This condition did not reappear in the next generation.

Genetics of Splitfoot

Matings of three males and four females produced both normal and splitfoot embryos (Table II, original matings). In backcross matings only one bird (2170) produced offspring which deviated significantly from a 3 normal: 1 splitfoot ratio. Because of the excess of splitfoot in the progeny of 2170, both the test mating subtotal and the grand total showed a significant deviation from expectation. However, no significant heterogeneity was revealed, within the test matings or within all matings combined, by an interaction chi-square test (Table III).

In both the original and test matings there was a slight but non-significant excess of males among splitfoot progeny and of females among normal progeny sexed (Table IV).

All the data indicate that splitfoot is due to homozygosity of an autosomal recessive gene which is lethal during terminal stages of embryonic development, at least in the highly inbred stock of its origin.

Compared with other mutations affecting the appendicular skeleton of the fowl, splitfoot differs genetically from polydactyly and duplicate polydactyly, which result from the action of single autosomal dominant genes. Like diplopodia and both talpids it results from homozygosity for a lethal autosomal recessive gene (suggested symbol *sf*).

In its least severe expression, elongated hallux, splitfoot resembles polyphalangism. It

TABLE III. Heterogeneity chi-square for test matings and all matings combined

	d.f.	χ^2
Test Matings:		
Total	8	23.95*
Pooled	1	7.33
Interaction	7	16.62
All Matings:		
Total	12	27.52*
Pooled	1	4.26
Interaction	11	23.26

*$P < .01$

TABLE IV. Sex ratio in splitfoot and normal progeny

	Splitfoot					Normal				
	Total	♂♂	♀♀	Not sexed	% ♂♂	Total	♂♂	♀♀	Not sexed	% ♂♂
Original matings	41	20	19	2	51.3	117	41	51	25	44.6
Test matings	47	25	19	3	56.8	86	40	44	2	47.6
Total	88	45	38	5	54.2	203	81	95	27	46.0

differs from the other mutants in that its expression forms a gradation from the pre-axial to the post-axial area of the posterior limb rather than being restricted to the region of the hallux.

If one considers splitfoot in the context of the five other mutants discussed here, it occupies an intermediate position, with respect to its range of effect, between the talpids which alter the entire limb plate and common polydactyly in which the hallux is present and may be either duplicated or elongated. In diplopodia the hallux is typically absent, but digits II, III and IV are invariably present and normal. In splitfoot only digits III and IV remain unaltered. All of these mutants are highly variable in phenotypic expression. While splitfoot is lethal, it is quite possible that some of the homozygotes might hatch in a noninbred stock. At present the splitfoot line is being out-crossed to several nonrelated stocks of chickens, including two diplopod-carrier lines differing in penetrance and expressivity of *dp*.

Summary

A new autosomal recessive lethal gene affecting the foot structure of the domestic fowl has been described. In its most typical phenotypic expression the second digit is duplicated and the hallux is absent. A great deal of phenotypic variation can be observed, ranging from an elongation of the hallux to a duplication and even a triplication of the second tarso-metatarsal bone and its associated digit. Homozygous *sfsf* embryos die between the seventeenth and nineteenth days of incubation. Splitfoot embryos also have a reduced premaxilla and an eye defect involving an incomplete eyelid sometimes accompanied by cataract. The albumen in many cases is not utilized by the embryos.

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Correction—Mouse Mutation Article

An error appeared in the sentence beginning at the bottom of column one and continuing to the top of column two, page 121 of the article by Isherwood *et al.*, "A New Mutation of *a* to a^t in the Mouse," which appeared in the May-June issue of the *Journal*. The sentence as it should read is as follows:

To test the validity of the impression that the new mutation was indeed black-and-tan modified by chinchilla genes at the *c* locus[3], mutant mice were crossed to mice of the C57BL/St (*aaBB*), BRS/St (*aabb*), C3H/St (*AABB*) and YBR/Wi/Ha (A^wabb) strains.

The editor regrets the error.

SECOND LIST OF KNOWN GENES IN THE TOMATO

Including Supplementary Rules for Nomenclature

C. D. Clayberg, L. Butler, C. M. Rick, and P. A. Young*

SINCE the compilation of the last list of known genes in the tomato by Barton *et al.*[1], 175 new mutants have been described. These, combined with the genes in the first list, bring the total to 282. The continued growth of interest in tomato genetics makes desirable the publication of a new list bringing the previous one up to date. Some symbols of the previous list also require revision as a consequence of tests for allelism and discovery of mimics.

At the same time there presently is a need for a clarification of the system proposed for naming and applying symbols to new mutants. The Gene List Committee of the Tomato Genetics Cooperative thus recommends that the following items, which conform to the recently adopted system of the International Committee on Genetic Nomenclature[18], be used as a supplement to the list of rules already published[1].

Part 3 of the original rules states: "Mutant genes are designated by letter symbols. The mutant name comprises an adjective or noun or combination of both that refers to the main diagnostic feature of the phenotype. The initial letter of the symbol should be the same as that of the name; additional appropriate letters are added as necessary to distinguish it from other symbols already in use."[1] Although this is a clear statement, it has not been adhered to in some recent instances, and amplification is needed to meet some newly arisen situations. We hope that before publication of a new symbol, the corresponding gene will be checked for allelism with other genes of similar phenotype, for duplication of previously existing symbols, and for conformity with the adopted rules.

The following rules have therefore been proposed to clarify tomato gene nomenclature. Adherence to them by all describing new mutants will greatly help to minimize confusion and to improve gene naming and symbolizing.

Additional Rules of Nomenclature

1. Special effort should be expended to find *appropriate* names for new mutants. Latin names or their English derivatives are recommended for their international meaning.

2. The symbol should consist of no more letters or numbers than necessary to distinguish it from all other existing symbols. Although lengthy symbols may have reference value to the discoverer, they are of no significance to other workers, who are often likely to make greater use of the mutants than the discoverer. When the length of the symbol approaches that of the name from which it was derived, it no longer is a symbol and becomes too cumbersome for genetic notation.

3. Symbols for mimics of a single series should not be composed of various permutations of the letters of the same name. If a name is adopted for a series of mimics, all members of that series should be designated by the same name and symbol with proper numerical subscripts.

4. Much confusion will be avoided

*Gene List Committee, Tomato Genetics Cooperative. Communications concerning this article and other affairs of the Tomato Genetics Cooperative should be directed to C. D. Clayberg, Chairman of the Gene List Committee, T.G.C., Department of Genetics, Connecticut Agricultural Experiment Station, New Haven; or to C. M. Rick, Chairman of the Coordinating Committee, T.G.C., Department of Vegetable Crops, University of California, Davis.

by clearing names and symbols with the Chairman of either the Gene List Committee or Coordinating Committee of the Tomato Genetics Cooperative at the earliest opportunity. Such action will settle matters of priority and assist the investigator by assigning a reasonably permanent symbol to his mutant. Permanence cannot be guaranteed, however, because unknown earlier or simultaneous publication might establish priority.

Second List of Known Mutant Genes

The following list contains symbols, descriptions, and references for 175 new mutant genes not included in the list of Barton *et al.*[1].

Symbols have been revised when tests have indicated allelism, symbol conflicts have occurred, or symbols have not followed the revised nomenclatural rules. Parentheses following the preferred gene symbol contain synonyms or former designations.

Symbol	Character	Reference
ac	*apocarpous.* Highly deformed multicarpellate and apocarpous fruits.	6
ae (a_{288})	*entirely anthocyaninless.* Lacks anthocyanin in the seedling stem.	20
acg	*aegrota.* Brown necrotic layer spreads over leaves, which die prematurely.	16
af (a_{288})	*anthocyanin-free.* Completely free of anthocyanin.	20
ah	*Hoffman's anthocyaninless.* Completely free of anthocyanin.	15
ai (a_{249})	*incomplete anthocyanin.* Early seedling stem has trace of anthocyanin, soon lost.	20
ang	*angustifolia.* Reduced, sometimes chlorotic, narrow leaves; slow growth.	17
ao (a_{287})	*anthocyanin omitted.* Lacks anthocyanin in the seedling stem.	20
ar	*arrecta.* Small, compact plants; younger leaves yellow-green, later normal color.	17
as_6	*asynaptic-6.* Delayed and asynaptic meiosis; chromatin degeneration; possibly two loci involved.	20
atn (*at*)	*attenuata.* Narrow, pointed leaves, yellow-green, purple below; rigid cylindrical habit; thin shoots.	17
au	*aurea.* Bright yellow foliage, often whitish; pale yellow corolla and stigma.	10
bc (*bi*)	*bicolor.* Young leaves mottled brownish olive normal, later glazed dark green; short internodes; heterozygote intermediate for some characters.	17
bg	Resistance of fruit to bursting.	20
bi	*bifurcate inflorescence.* With *f* and *j* causes extreme fasciation of the "ring type."	12
bl	*blind.* Stem terminates in first inflorescence; midribs may develop adventitious shoots.	14
bn	*blunt.* Variable stamenless, usually with petaloid stamens adnate to pistil; leaf segments obtuse.	20
Bt	Susceptibility of fruit to bursting.	20
btl	*brittle.* Growth ceases with first flowering; progressive defoliation; temperature-sensitive.	20
bu^{hem} (fru^{hem})	$bushy^{hemiglobosa}$. Phenotype like *bu* except leaves longer, more lax; heterozygote intermediate in some traits.	17
bul	*bullata.* Leaf surface blistered, chlorotic with network of darker veins; short internodes.	17
car	*carinata.* Shortened, keeled pinnae, dark gray-green color; deformed flowers; elongate fruits.	16
cla	*clara.* Yellowish-green leaf color, purple veins and petioles; incompletely dominant.	16
clau	*clausa.* Cleistogamous; leaf lamina foreshortened, excessively divided, long-petioled; flattened fruits.	17
cm	*curly mottled.* Strong virus-like mottling and distortion of leaves and abortion of flowers; highly sensitive to environment.	14

Symbol	Character	Reference
cn (*ca*)	*cana.* Cotyledons and leaves gray-green especially on undersides; tiny, unbranched plants.	17
co	*cochlearis.* Reduced gray-green leaves with concave spoon-shaped leaflets; small plants of normal habit.	17
con	*convalescens.* Yellow-green, paler at growing point; variably depressed growth.	16
cr	Resistance of fruit to radial cracking.	20
cs	*corollaless.* Corolla and androecium sepaloid, stigma irregular and protruding.	20
Cu	*Curl.* Mid- and lateral veins and petiole greatly foreshortened producing crumpled leaf; homozygous viable.	22
cv (*cu*)	*curvata.* Young stem curves from node to node; dark rugose leaves; dwarf compact habit; heterozygote intermediate for some traits.	17
$d_1^{cr}(rob^{cris})$	*dwarf-1*^{crispata}. Allele of phenotype intermediate between d_1 and d_1^{cr}.	16
d_2	*dwarf-2.* Slow-growing plant with normal stem and leaf proportions; semi-sterile; classification good when three weeks old.	20
de	*declinata.* Great reduction in size of all parts; leaves dark yellow-green, leaflets roll toward underside.	17
deb	*debilis.* Leaves emerge yellowish; necrotic mottling, whitish, later brown, often constricting leaves in middle.	16
def	*deformis.* Seedlings normal, later leaves becoming progressively reduced, filiform, with adnate segments; deformed flowers and reduced fertility.	16
deli	*deliquescens.* Early habit dense, later loosely parting; shortened yellowish leaves with narrow acute segments.	17
dep	*deprimata.* Slightly depressed growth; dainty, darker leaves.	16
depa	*depauperata.* Very small plants with short internodes; leaflets bowed, color variable, mostly yellowish.	17
di	*divergens.* Rachis very short, leaf color yellowish gray-green.	17
dil	*diluta.* Dull light green, small, rough leaves; plants half normal size.	16
dim	*diminuta.* Somewhat smaller plants and leaves, shortened internodes; older leaves gray-green with violet veins.	17
dis	*discolor.* Leaf color light green with virescent growing regions, darker veins.	17
ds	*dwarf sterile.* Stunted plants with short internodes and reduced leaves; partially male-sterile.	20
ec	*exserted carpels.* Fruits vary from none to many exserted carpels.	20
elc	*elegans.* Leaves small and dainty, yellow-green; tiny plants.	16
elu	*eluta.* Yellow-green leaves; strong branching tendency; slight growth depression.	16
er	*erecta.* Dwarf bushy plants, short internodes; shortened dark green leaves; heterozygotes intermediate.	17
exl (*ex*)	*exilis.* Very small plant; tiny plicate leaves of light gray-green color.	17
ff	*filicifolium.* Leaves 3- and 4-pinnately divided, with acute segment tips.	15
fir	*firma.* Broad, blunt leaf segments; bushy cylindrical plants; foreshortened inflorescence.	17
fla	*flavescens.* Light green leaves with fewer segments; variably reduced plant size.	16
flav	*flavida.* Leaves yellowish, mottled yellowish green, small; heterozygote intermediate for some traits.	17

Symbol	Character	Reference
fu	*fusiformis.* Early growth habit bushy, spindle-shaped; leaves shortened, curved upwards, shiny yellowish, paler at growing point.	17
ful	*fulgens.* Yellow at growing point, older leaves becoming greener; golden yellow in field; yellow unripe fruit color.	16
Fw	*Furrowed.* Deeply furrowed, dark green cotyledons; severely stunted plant; homozygous inviable.	14
gf	*green flesh.* Persistent chlorophyll giving ripe fruit purplish-brown color.	20
gh (ab)	*ghost.* Incomplete chlorophyll deficiency, starting green, later breaking to white.	20
gil	*gilva.* Yellow cotyledons; highly variable ratios.	20
glau	*glaucescens.* Leaves shortened, dull green to yellowish gray-green; small upright habit.	17
glo	*globosa.* Short internodes and leaves, pale green color; incompletely dominant.	16
gq	*grotesque.* Irregular androecium; pistil variably twisted, elongated, ridged, or lobed.	14
gr	*green ripe.* Resembles *gf*, except that center of fruit turns red.	20
gra	*gracilis.* Very small, unbranched, delicate plants; much reduced leaves yellowing at margins.	17
$hp_1 hp_2$ (*bs,dr*)	*high pigment-1, -2.* Intensify chlorophyll, carotenoids, ascorbic acid of fruit.	19
ht	*hastate.* Primary and mature leaves elongate, more entire, fewer lobed; abortive and deformed flowers.	15
ig	*ignava.* Dwarf upright, little branched; leaves relatively large, light green.	17
imb	*imbecilla.* Weak plant with few branches; younger leaves bright yellow-green, older light green.	16
in	*indiga.* Small, dainty, nearly unbranched plants; shortened light gray-green leaves.	17
inc	*incurva.* Leaf segments weakly curved, undulate margins; stems, leaf veins, and inflorescence crooked.	17
inf	*infirma.* Tiny plant with very short internodes; leaves small, plicate, strongly down-curved.	16
int	*integerrima.* Leaflets almost entire, terminal enlarged, yellowish color.	17
intro	*introflexa.* Leaves tend to roll toward upper side, dull gray-light green; shortened internodes; excessively branched.	17
inv	*invalida.* Small, dainty plants; irregular chlorotic flecks, later leaves deformed.	16
irr	*irregularis.* Shortened leaves, irregularly veined, darker green, yellow tinged.	17
j_2	*jointless-2.* Jointless elongate pedicel; teratological calyx; proliferated inflorescence; elongate fruit.	13
jug	*jugata.* Fasciated stems and fruits; short internodes; inflorescence almost sessile.	16
l_2	*lutescent-2.* Premature yellowing of older leaves; yellowish unripe fruit.	20
La	*Lanceolate.* Simple, entire, elongate small leaves; slender stems, excessively branched; small fruits; homozygous inviable.	20
lat	*lata.* Habit broad at first; growth much depressed in field.	16
lo	Reduced locule number.	20
ls	*lateral suppressor.* Few or no axillary branches; suppressed corolla; partially male-sterile.	20
luc	*lucida.* Leaves bright green, yellow-green at growing point.	16
lut	*lutea.* Leaves yellow-green with darker veins; growing point much yellower.	16

Symbol	Character	Reference
M_3 (M_{3d})	*Mottled-3.* Variable sectoring for chlorophyll deficiency; homozygous lethal.	20
marm	*marmorata.* Marbling of several tones of white-light green on leaves, strongest expression in greenhouse.	16
md (*mo*)	*mottled-2.* Many small chlorotic spots on leaves; temperature-dependent.	20
Me	*Mouse ears.* Leaves 3- to 4-pinnately compound with reduced clavate segments; shortened internodes; homozygous viable; incompletely dominant.	15
Mi	*Meloidogyne incognita.* High-level resistance to the nematode, *M. incognita.*	5
mn (*mi*)	*minuta.* Small, upright, almost unbranched plants; shortened leaves prematurely yellowing.	17
mo_n (i^B)	modifier of *B.* Increases content of beta carotene in presence of *B.*	21
mon	*monstrosa.* Very tiny, upright, unbranched plants; leaves much reduced, down-curved, ball-shaped; heterozygote intermediate for some traits.	17
ms_{19}	*male-sterile-19.* Slightly shrunken anthers; stigma mostly protruding; variety Garden State.	20
ms_{20}	*male-sterile-20.* Anther tips equal or exceed stigma; variety Garden State.	20
ms_{21}	*male-sterile-21.* Anthers and style of equal length; flowers slightly smaller; variety Rutgers.	20
ms_{22}	*male-sterile-22.* Anthers normal length, shrunken; flower color slightly lighter; variety Garden State.	20
ms_{23}	*male-sterile-23.* Anthers slender and paler; no pollen; meiosis delayed and prolonged; stigmas depressed; variety Earlypak.	20
ms_{24}	*male-sterile-24.* Anthers slightly more slender and paler; 100 percent aborted pollen; meiosis normal; stigmas mostly exposed; variety Earlypak.	20
mult	*multiflora.* Very greatly increased number of flowers per inflorescence.	16
na	*nana.* Tiny plant with very short leaves and internodes; leaves plicate, rugose.	16
nd	*netted.* Primary leaves attenuated and chlorotic; chlorophyll concentrated around veins of later leaves.	20
neg	*neglecta.* Small, weakly branched plants; paler leaves, with darker veins, becoming progressively necrotic, prematurely dropping.	16
ni	*nitida.* Leaves long-petioled with dainty, deeply cut segments.	17
not	*notabilis.* Dainty, delicate, yellow-green leaves; tends to wilt in dry, hot weather.	17
Nr	*Never ripe.* Fruits ripen slowly to a dirty orange color; homozygous viable.	14
nv	*netted virescent.* Pale interveinal areas of cotyledons and true leaves; older leaves never with full green color.	20
op	*opaca.* Lighter leaf color, yellow at growing point.	17
pal	*pallida.* Light green color; paler corolla; plant size variable; foliage color dominant.	16
pau	*pauper.* Tiny, weak, unbranched plants, tending to die before fruiting; marked reciprocal graft influence with normal.	17
pc	*precocious centromere division.* Centromeres divide prematurely in meiosis starting in anaphase I and completed in all by prophase II; completely pollen-sterile, highly egg-sterile.	2

Symbol	Character	Reference
per	*perviridis.* Leaves darker green, dropping early, anthocyanin strong; very small, slow-growing plants.	16
pg (*pg_{3a9}*)	*pale-green.* Cotyledons first pale yellow, turn gray-green, which is mature plant color; slow-growing.	20
Pi_{T1} (*TR_1*)	Susceptibility to *Phytophthora infestans* race T_1; resistance to race T_0.	4
pic	*picta.* Leaves at first normal green, later turning yellowish, then brown necrotic above, some eventually brown below.	17
pro	*procera.* More rapid growth rate; few foliolules, larger segments entire.	16
prun	*prunoidea.* All parts elongate, particularly the fruit; traits weakly dominant.	16
pt	*petite.* Small, slow-growing chlorotic plants; light green cotyledons.	20
pul	*pulvinata.* Shortened internodes, stronger branching; elongate cotyledons; leaflets broad and much divided.	17
pum	*pumila.* Tiny, dainty plant with few or no branches; slow growth rate.	16
pur	*purilla.* Plants much retarded, erect, little branched; leaf edges yellow with normal green veins.	16
ry (*ry*)	*reddish yellow.* Likely allele of *r* with reddish flesh tones in ripe fruit.	20
ra	*rava.* Down-curled, dull gray-green leaves; more hairy than normal.	16
re	*reptans.* Seedlings inclined from epicotyl, mature plants recumbent; elongate internodes; less branched.	17
rela	*relaxata.* Lax open habit; tiny plants; light green, yellow-tinged leaves; heterozygotes intermediate color.	17
rep	*repens.* Strong creeping tendency even in seedling; less branching; shortened, prematurely dying leaves.	17
rl (*ra*)	Resistance of fruit to radial cracking.	20
rob_2 (*rob* II)	*robusta-2.* Shortened internodes and leaves; thick, rugose, bluish gray-green leaves; not allelic with the mimic, d_1.	17
ru	*ruptilis.* Leaflets narrow, keeled, dull light green, darker veined; heterozygote temporarily intermediate.	17
rust	*rustica.* Dwarf plants with short internodes; leaves broad, blunt with fewer segments.	16
sd	*sun dwarf.* Very short, scarred internodes in high light intensity; heterozygotes usually intermediate.	14
sf	*solanifolia.* Primary leaves entire; segments of later leaves entire and folded; filiform calyx and corolla.	20
sit	*sitiens.* Smaller plant, very weak in field; very short leaves, down-curled, becoming necrotic and dropping prematurely.	16
sl^2 (*sl_2*)	*stamenless-2.* Nearly stamenless, anthers much distorted; *sl^2* to *sl^5* likely alleles of *sl^1*.	7
sl^3 (*sl_3*)	*stamenless-3.* Like *sl^2* except for greater anther development.	20
sl^4 (*sl_4*)	*stamenless-4.* Like *sl^3* except anthers nearly normal in winter greenhouse.	20
sl^5 (*sl_5*)	*stamenless-5.* No anthers; sepal-like petals.	20
squa	*squarrosa.* Small, weak plants of squarrose habit, few branches; small, pale gray-green leaves; heterozygote intermediate in color and size.	17
stri	*stricta.* Rigid upright growth; broader blistered leaves.	16
su	*suffulta.* Leaves deeply veined, yellow-tinged; fruit cluster upright; strong anthocyanin.	17
sub	*subtilis.* Tiny plant with short internodes, fastigiate habit; narrow acute leaf segments.	16
suf	*sufflava.* Uniform light green color.	16

Symbol	Character	Reference
sulf$^{pura\ 1\text{-}10}$	*sulfurea*$^{pura\ 1\text{-}10}$. Series of 10 alleles with cotyledons greenish yellow becoming pale yellow, true leaves pure yellow; lethal unless grafted on normal stock.	8, 20
sulf$^{vag\ 1\text{-}3}$	*sulfurea*^{variegata}. True leaves variegated green-white; some homozygotes lethal unless grafted; three alleles.	8, 20
Sus	*Subsistens*. Growth variably suppressed; few or no branches; varied meiotic abnormalities; homozygous lethal.	20
Swf^{a}	*Spotted wilt resistance-1*^{a}. Resistance to strains TB_2, N_1, and R_1; variety Pearl Harbor.	3
Swf^{b}	*Spotted wilt resistance-1*^{b}. Resistance to strain TB_2; variety Rey de los Tempranos.	3
sw_2	*spotted wilt resistance-2*. Resistance to strains TB_1, N_2, R_2, and R_3; variety Rey de los Tempranos.	3
sw_3	*spotted wilt resistance-3*. Resistance to strains R_2, R_3, and M_2; variety Rey de los Tempranos.	3
sw_4	*spotted wilt resistance-4*. Resistance to strains M_1, M_2; variety Rey de los Tempranos.	3
sy	*sunny*. Cotyledons green bleaching to yellow; young leaves yellow at base, turn green.	20
t^{v}	*virescent tangerine*. Fruit and flower color typical of *t*; irregular yellowing near growing point.	20
tab	*tabescens*. Normal seedling growth followed by severe stunting; leaves irregular, yellow-green, violet veined with necrotic specklings.	17
ten	*tenuis*. Very slow growth; leaves light green, reticulated with whitish yellow veins.	16
Tm_1	*Tobacco-mosaic virus resistance-1*. Moderate-level resistance.	9
Tm_2	*Tobacco-mosaic virus resistance-2*. High-level resistance.	20
um	*umbrosa*. Mature leaves darker green with wilted appearance; later growth stunted.	17
uni	*unicaulis*. Weak, thin, upright growth with great suppression of branching.	16
va^{dec}	*varia*^{decolorata}. Bright yellow-green at growing point, older leaves becoming normal green; dominant to *va*^{virg}.	16
va^{virg}	*varia*^{virgata}. Like *va*^{dec} except older leaves retain yellow in region of veins.	16
var	*variabilis*. Leaves light green fading to yellow-green at growing point; slightly smaller plant with shorter internodes.	16
vel	*velutina*. Light yellow-green leaves, paler at growing point, which has velvety appearance.	17
ven	*venosa*. Variably reduced growth; tiny folded leaves, whitish yellow with green veins.	16
ver	*versicolor*. Younger leaves with fine mottling of yellow; green veins.	17
vio	*violacea*. Heavy anthocyanin on stems and veins; dull light green leaf color.	17
vir	*viridis*. Early leaves dark green, third order leaf divisions.	16
vit	*vitiosa*. Leaves become progressively more deformed with twisted, filiform leaflets.	17
w_2	*wiry-2*. More extreme strap-shaped leaves, petals, and sepals than w_1, plant sterile.	11
w_3 (w_2)	*wiry-3*. Resembles w_1, except ovary more syncarpous.	20
Wo^{m}	*Morgan's wooly*. Pelage on all parts wooly but less than *Wo*; homozygous viable; heterozygote intermediate.	14
Wo^{v}	*vanWert wooly*. Denser hairiness than *Wo*; homozygous lethal.	20
yg_1	*yellow-green-1*. All foliage yellow-green, lighter near growing point.	20

Symbol	Character	Reference
yg_2 (yg_{2nn})	*yellow-green-2.* Foliage chlorotic yellow-green under all conditions; long hypocotyls.	20
yg_3 (yg_{3nn})	*yellow-green-3.* Resembles yg_4 with leaves appearing ragged.	20
yg_4 (yg_{4nn})	*yellow-green-4.* Foliage chlorotic yellow-green under all conditions; leaves more deeply serrate, edges curled.	20

Symbol Revisions for Genes of Previous List[1]

Former symbol	Preferred symbol
a	a_1
an	an^1
ca	an^2
d, rob^{mm}	d_1
d^z	d_1^z
j, lf	j_1
l, rab	l_1
m	m_1
ol	O^l
sl	sl^1
w	w_1

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INHERITANCE OF BRACT COLOR IN POINSETTIA

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THE bracts of most wild poinsettias (*Euphorbia pulcherrima* Willd.) are red, but plants with white bracts were collected as early as 1835 in Mexico[2]. The modern cultivars include plants with pink as well as those with red or white bracts. The origin of "pink" cultivars is unexplained, and there are no reports of the collection of plants with pink bracts from the wild.

A seedling with pink bracts was described by Adnet[1], who obtained it from a cross of red × white. Robinson and Darrow[6] refer to the origin in a German nursery of a pink form as a bud sport from a white plant.

A Missouri Botanical Garden Bulletin[2] reported the results of all possible self- and cross-pollinations of the red, white, and pink cultivars available in 1925. No seeds were obtained from self-pollination and only four crosses were successful; each produced one fruit and apparently only one seed in each fruit. Red × red gave a red seedling, red × pink gave pink, pink × red gave red, and pink × white gave pink. "Pink Ecke" was then in the commercial trade and probably was the pink cultivar used.

Percy-Lancaster[5] described the cultivar "Pink Beauty" and stated that it originated from a cross of red × white. He reported that numerous attempts to obtain seeds failed until 1934 when three seeds were obtained on a red seed parent. Two of these seeds germinated and one grew into a plant with red bracts and the other into a plant with white bracts.

Ewart[4] in 1956 stated that segregation for bract color occurred in progenies of red and white types and no true pinks were observed, but that one white type showed a tinge of pink in the bracts.

Until the last four years little poinsettia breeding has been carried on in the United States. New varieties have originated as sports or somatic mutations in the large numbers of the asexually propagated commercial crop. The Ecke family of Encinitas, California, have selected, named, and introduced nearly every one of the commercial cultivars in the past 40 years. Mr. Paul Ecke has drawn up a family tree of the modern cultivars which indicates that the cultivar "Oakleaf," a seedling grown in 1923, through sporting gave rise to "Ruth Ecke," "Albert Ecke," "Improved Albert Ecke," "Barbara Ecke Supreme," "Mrs. Ruth Ecke," "Indianapolis Red," "Improved Indianapolis Red," and "Dark Indianapolis Red." These varieties are red and include every red cultivar of economic importance. "Ecke White" originated as a seedling in 1945, but "Pink Ecke" and "Early Brick Red" existed in 1915, when Mr. Ecke's records begin. "Early Brick Red"

TABLE I. Inheritance of bract color in progeny of crosses involving the poinsettia cultivars "Ruth Ecke" and "White Ecke"

Pollinations	Bract color of seedlings	
	Red	White
Ruth Ecke selfed	51	0
Ruth Ecke F_2	70	0
White Ecke selfed	0	59
(White Ecke selfed) × White Ecke	0	78
White Ecke F_2	0	115
White Ecke × Ruth Ecke	151	1*
(White Ecke × Ruth Ecke) × Ruth Ecke	236	2*
(White Ecke × Ruth Ecke) × White Ecke	40	37
(White Ecke × Ruth Ecke) F_2	266	96

*Probably seedlings from self-pollinations in the unemasculated flower heads of the female parents.

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sported to "Hollywood," a red form, and to "Early White." "Pink Ecke" has very frequently given rise to red shoots, all apparently identical and called "St. Louis," which in turn has been recorded as sporting back to pink. "Santa Barbara" is a red cultivar of separate but uncertain origin in 1937. A 1957 introduction, thought by Ecke to be of seedling origin, is a cultivar called "New Pink."

Thus the literature presents a confusing and contradicting account of the inheritance of bract color in poinsettia. When a program of breeding for leaf-holding ability in poinsettia was begun at Beltsville, the cultivar "White Ecke" was found to be the principal source of this characteristic. Many populations from crosses of white, red, and pink parentage have been flowered, and information on the inheritance of bract color is presented.

Materials and Methods

Stock plants of the named cultivars were originally donated by Mr. Ecke. Collections of cultivars and native material have been received from the New Crops Research Branch. The present report is based upon crosses made and seedlings grown in the greenhouses at the Plant Industry Station, Beltsville. Because all crossing was done in December and January, protection from insect pollination was not necessary. Since emasculation is very difficult, crosses were made using the terminal female flower in cyathia located near the edge of the cluster where the male flowers either were late in emerging from the involucre or did not appear at all. Data from many crosses indicated approximately one percent contamination in the form of self-pollinations. Temperatures were kept at or above 70° F. during the crossing season. Crosses made in December and January produced fruits that matured in April and May, and seeds planted in June and July produced plants which flowered in December. Seeds were fully matured at harvest and germinated within two weeks after planting.

Results

A series of testcrosses between the cultivars "Ruth Ecke" and "White Ecke" (Table I) indicated that white bracts differ from red bracts by a single recessive factor, designated *wh*. The red factor (*WH*) is completely dominant over white. Both cultivars are homozygous for bract color. Data from reciprocal crosses were combined in Table I because no differences were ever observed. Two small backcross populations [(White Ecke × Ruth Ecke) × Ruth Ecke] were tested by crossing each plant to white (*whwh*). Nearly half, 11 of 25, gave half red seedlings and half white ones and were thus shown to be heterozygous for white.

Data from a few testcrosses of other cultivars are presented in Table II. "Early White" crossed to "Ecke White" gave all white seedlings. A new white plant (P.I. 245349) has also given all white seedlings in crosses to these white cultivars. All the other cultivars were apparently homozygous (*WHWH*).

Eleven new individuals with red bracts collected in Mexico (P.I. 245350 B through L) are apparently also homozygous for *WH*. The seedlings from the testcrosses have not flowered as yet but they have shown red pigment in the stem and petiole, a pleiotropic effect of the *WH* gene.

While the difference between red and white bracts is due to a single factor,

TABLE II. Inheritance of bract color in poinsettia cultivars

Cultivar	Bract color	Number of gametes tested	Genotype *wh*	Genotype *WH*
Early White	white	30	30	0
Pink Ecke	pink	25	0	25
New Pink	pink	34	0	34
Mexican Pinks*	pink	7	0	7
St. Louis	red	13	0	13
Santa Barbara	red	11	0	11
Oakleaf	red	28	0	28
Tetraploids†	red	117	0	117

*Two pink clones introduced as PI 245347 and 245345.

†This group includes five different cultivars all derived from "Oakleaf" by either spontaneous or colchicine-induced chromosome doubling.

modifying factors affect each of these phenotypes. The red bracts vary from a blue red to an orange red and from bright, clear red to dull, gray red. The white seedlings vary from greenish and creamy whites to pure, clear white. Approximately 3,500 seedlings have been flowered and all had either red or white bracts. About 350 have been first- or second-generation descendants of plants with pink bracts and all of these have also possessed red or white bracts.

Discussion

All the cultivars with red bracts that were tested proved to be homozygous for the gene *WH*. However, only three different genotypes were represented, since "Ruth Ecke" and the tetraploids have apparently all arisen by somatic mutation or chromosome doubling from the cultivar "Oakleaf." Their genetic similarity would support Ecke's statement that their origin was by somatic mutation. The apparent homozygous nature with respect to red bract color of the 11 new red acquisitions from Mexico would indicate the widespread occurrence of the *WH* gene in the native populations as well as in the commercial cultivars. While there are several reports of white poinsettias in the wild, none have been found recently. The exact origin of the three white clones available for this study cannot be determined. Even though apparently from widely different sources, all are determined by the same *wh* allele.

The fact that the cultivar "Pink Ecke" is genetically red is not surprising in view of its periclinal nature as reported by Bergann[3]. This would indicate that the pink poinsettia chimera described by Robinson and Darrow[6] was also "Pink Ecke." The frequent sports to "St. Louis" from "Pink Ecke" which are found in every commercial range therefore are not sports, but the exposure of the internal layers of the chimera in adventitious buds.

The cultivar "New Pink" and five different P. I. introductions from Mexico which had pink bracts have all been very similar in appearance and growth responses. Only stems with red bracts have been produced from adventitious buds on root cuttings. "New Pink" and the two Mexican pinks which produced seeds were genetically red. It is, therefore, suggested that all of these are part of the same clone which makes up the cultivar "Pink Ecke."

All evidence from the present study is that the pink bracts are due to a mutation that is not present in the germ line in any material now available and that they are not a result of the heterozygous condition of the known alleles at the *wh* locus. The earlier reports of pink seedlings could be explained as different terminology in description of the colors involved or by the possibility that the genetic basis of color inheritance was entirely different in the material used by the earlier workers.

Summary

With respect to bract color, poinsettias with white bracts differ from the normal red by a single recessive factor designated *wh*. All commercial cultivars with red bracts bred true for red bracts, as did "Pink Ecke" and "New Pink." No pink seedlings have appeared even in descendants of crosses involving the pink parents. "Ecke White" and "Early White" gave all white-bracted seedlings from both self- and cross-pollinations of these two varieties.

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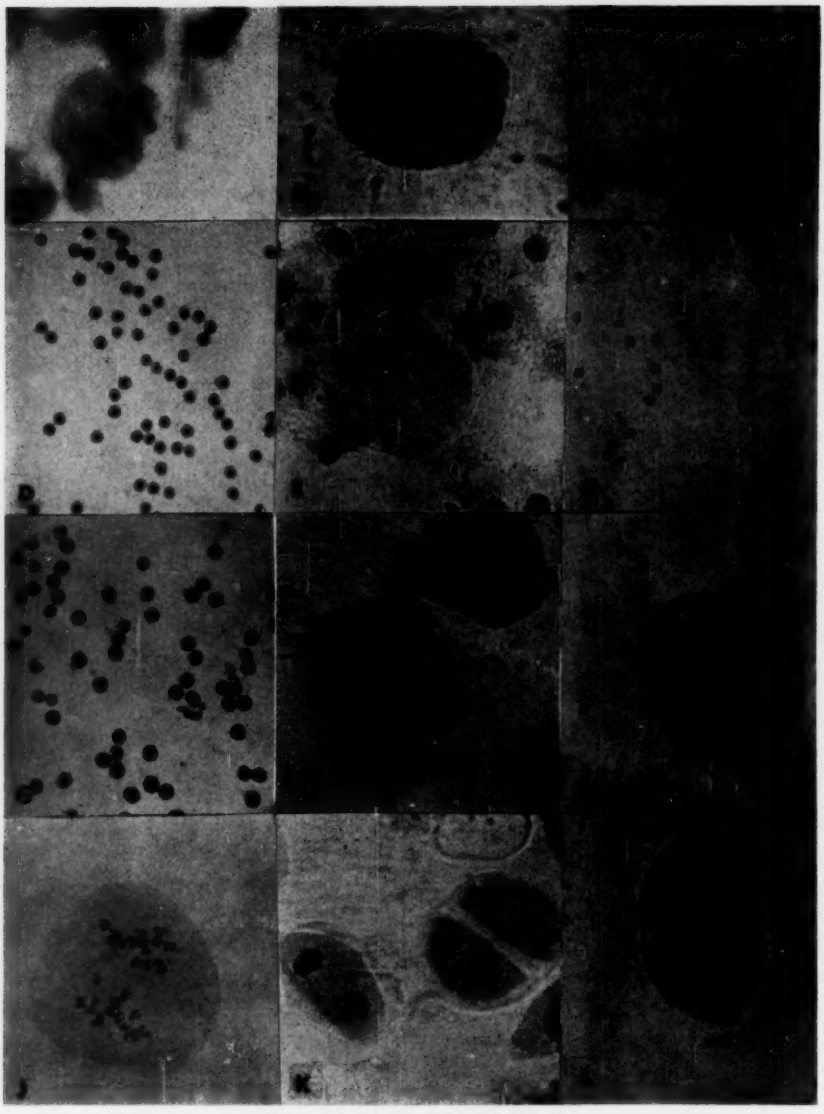

POLLEN AND MEIOTIC BEHAVIOR

Figure 6

A—Chromosomes of *H. pusillum*. *B*—Chromosomes of *H. compressum*. *C*—Pollen of *H. pusillum*. *D*—Pollen of *H. compressum*. *E*—Abnormal chromosome pairing of the F_1 hybrid. *F*—Pollen sterility of F_1 hybrid. *G*—Pollen of the F_2 allotetraploid. *H*—Diakinesis of F_2 allotetraploid. *I*—Metaphase I of the F_2 allotetraploid. *J*—Anaphase I of the F_2 allotetraploid. *K*—Dyad formation of F_2 allotetraploid. *L*—Tetrad formation of the F_2 allotetraploid.

WILD BARLEY HYBRIDS

I. *Hordeum compressum* × *Hordeum pusillum*

A. B. SCHOOLER*

AN interspecific crossing program was initiated in an attempt to transfer the type of spot blotch resistance found in some wild *Hordeum* species to *H. vulgare*. Several research workers[1-4] have reported crossing studies among wild *Hordeum* species and other genera. One cross between *H. leporinum* × *H. vulgare* has been reported[3], but in most instances, crosses including wild *Hordeum* species × *H. vulgare* have been without success.

Most wild *Hordeum* species do not cross readily with cultivated barley. However, it is possible that interspecific hybrids between the wild species will cross more readily with *H. vulgare*. Therefore, our approach includes an attempt to produce interspecific hybrids between wild *Hordeum* species for eventual crossing and the transfer of disease resistance into cultivated barley.

Materials and Methods

Two wild species of *Hordeum*, No. 56-31 (*H. compressum*) and No. 528 (*H. pusillum*), were obtained from Dr. G. A. Wiebe, U.S.D.A., Beltsville, Maryland. They are highly resistant, though not completely immune, to attack by the spot blotch organism, *Helminthosporium sativum*.

Crosses were made between these two species in the greenhouse during the spring of 1957. Two of the resulting hybrids were crown-treated with a 0.02 percent solution of colchicine in an attempt to induce chromosome doubling. Cytological studies were conducted on pollen-mother-cells for determination of chromosome behavior of the parents, untreated F_1 plants, and the F_2 plants derived from seed of colchicine-treated hybrid plants. Pollen development was also studied by use of the potassium iodide technique with the above material.

Results

F_1 hybrids from crosses between the wild species No. 528 (*H. pusillum*) and No. 56-31 (*H. compressum*) were obtained both by embryo culture transfers shortly after pollination and by seed formation. Generally, the hybrid seeds produced from this cross were slightly shriveled but would germinate and produce F_1 plants. The F_1 plants (Figure 7), however, were completely sterile.

Two F_1 plants that were crown-treated with colchicine produced 37 viable seeds. Twenty of the above 37 seeds germinated and were grown in 6-inch pots in the greenhouse. No segregation was noted in the F_2 progenies (Figure 7) and all F_2 plants produced seed. Several seeds from the F_2 plants were planted in 6-inch pots and produced plants similar to the F_2 parents. The above plants are shown in Figure 7. The seed and spikes of the F_2 plants are larger than those of either parent.

Cytological examinations showed that each of the parent species has seven pairs of chromosomes (Figure 6*A* and *B*) and produces normal pollen (Figure 6*C* and *D*). The anthers of *H. pusillum* are very small and apparently contain very few pollen grains (Figure 6*D*). Further, examinations of pollen-mother-cells from the F_1 plants indicated that abnormal pairing existed between chro-

*Department of Agronomy, North Dakota Agricultural College, State College Station, Fargo, North Dakota. Published with the permission of the Director of the North Dakota Agricultural Experiment Station as journal article No. 5. The author gratefully acknowledges the support of the Malting Barley Improvement Association.

PARENT AND HYBRID PLANTS

Figure 7

From left to right are shown *H. pusillum* (528), F_1 hybrid, F_2 allotetraploid, and *H. compressum* (56-31).

mosomes of the two parents. The photograph of chromosome behavior in the F_1 plants (Figure 6*E*) shows loose pairing between three chromosomes. However, other observations of microspores indicated that loose pairing may occur between as many as five chromosomes, but very little or no chromosome pairing occurs in the majority of microspores. This lack of chromosome pairing probably accounts for the pollen sterility found in the untreated F_1 plants (Figure 6*F*).

The F_2 plants appeared to be very similar to the F_1 plants but were slightly larger and highly fertile (Figure 7). Several stages of microsporogenesis in the F_2 are shown in Figure 6*G-L*.

Although fertility appeared to be completely restored in the F_2 plants derived from the colchicine-treated F_1 hybrids, cytological examinations of pollen-mother-cells indicated that some chromosomal aberrations had occurred during the first division. This was evidenced by the fact that some chromosome fragments

were observed at the plate following anaphase I.

Discussion

On the basis of the results of this study it was shown that F_1 hybrids were obtained between the wild *Hordeum* species *H. compressum* and *H. pusillum*. The very loose pairing of chromosomes of the F_1 plants is explainable by the nonhomology of the parental chromosomes. The normal pairing of chromosomes in the F_2, which had a $4n$ chromosome complement, probably indicates pairing between parental chromosomes. The diploid type of pairing in the F_2 further suggests that an allotetraploid was produced from this cross.

The fact that no segregation occurred in the F_2 progenies was further evidence of the allotetraploid nature of the F_2 plants. If the parental chromosomes were doubled by colchicine treatment, the chromosomes would be homologous for the parental genomes and segregation would not be expected.

Summary

Allotetraploid plants were produced by doubling the chromosomes of the F_1 plants from a cross between the wild species *H. compressum* and *H. pusillum* by crown treatment with a 0.02 percent solution of colchicine. The F_2 and F_3 allotetraploid plants were highly fertile.

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FACTORS INVOLVED IN THE MECHANISM OF "TASTE-BLINDNESS"

ROLAND FISCHER AND FRANCES GRIFFIN*

TASTE sensitivity to phenylthiourea, also called phenylthiocarbamide (PTC), and to other structurally related anti-thyroid compounds has been shown to follow a bimodal distribution[7, 11]. Recent data[10] confirm the 7:3 ratio of tasters to non-tasters in a Caucasian population of adults and are in agreement with the genetic hypothesis that non-tasting is a single recessive character.

It has also been reported[3] that it is the individual's own saliva that is necessary for tasting PTC and related compounds. The nature of this "saliva factor," however, was not elucidated. Fawcett and Kirkwood[5] have presented evidence as to the presence of a soluble enzyme system, tyrosine iodinase,† in the parotid and submaxillary glands, and asked emphatically: "What on earth is this enzyme system doing in the salivary glands?"[9] However, a connection between thyroid metabolites and the "saliva factor" was not suspected.

Recently, while confirming the existence of the disputed[2, 4] "saliva factor" (to be published), we were fortunate in obtaining (through the courteous assistance of Dr. S. Garn, Yellow Springs, Ohio) a paper by N. Turner *et al.*[12] reporting protein bound iodine (PBI) and total iodine values in the saliva of 30 children 8 to 14 years old from among the school population brought to the Forsyth Dental Infirmary for Children for dental examination and treatment. When analyzing the distribution of free iodine (which we obtained by deducting the PBI from the total iodine for each child) in the saliva of Turner's subjects, we found that it resembled that of taste-blindness to PTC and related compounds measured in young individuals[8]. It is probable that the resemblance would be more striking if Turner's subjects were a random sample rather than a "dental" population and if the age groups of both populations were exactly matched, the reason for this being that taste threshold for PTC is apparently distributed on a continuum at birth and becomes only gradually bimodal during the growth process. Moreover, an examination of the data of Wolfe and Turner[13] shows that the amount of salivary peroxidase in 4- to 15-year-old individuals is inversely related to the amount of salivary free iodine obtained from the data of Turner[12].

The above conclusions gain significance if interpreted in the light of our following experiments. A 20-minute sample of mixed rubber-band-stimulated morning saliva (minimum volume 8 cc.) was centrifuged at +4° C. for 30 minutes at 3000 r.p.m. The supernatant was re-centrifuged and 1-ml. portions of it were diluted 1:5 with Tris buffer to pH 7.36 at 23° C. and supplemented with 6-n-propylthiouracil (PROP); final concentrations of Tris and PROP are 0.05 M and 3.75×10^{-5} M. Subsequently, the rate of reaction in a sample

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†Which needs cupric ion and tyrosine to synthesize free monoiodotyrosine; the system also requires hydrogen peroxide for the peroxidation of iodide[1].

was followed in a Perkin-Elmer "Spectracord 4000-A" spectrophotometer at 17°, 27° and 37° C. in the ultraviolet range. The decrease in optical density was read at the λ maximum between 276 and 284 mμ in 8 minutes. This decrease, which is not photosensitive, indicates a significantly faster rate of reaction for extreme "non-tasters" than for "tasters."

The "ΔO.D. 8-minute" effect can be simulated in the absence of saliva by the action of hydrogen peroxide on 6-n-propylthiouracil. The decrease in optical density thus produced, however, increases with increasing temperature, which effect is converse to that obtained with saliva.

Cellulose (standard grade, ashless, acid washed, Whatman), dialysis tubing ("Visking," No. 8-667 from Fisher Science Co.), the silica lattice of glass and electrolytic desalting (with a "RSCo" Electric Desalter, Model 1930 from Research Specialties Co.) are individually effective in partially or completely removing the "ΔO.D. 8-minute" effect from saliva. Polyvinylpyrrolidone ("Plasdone" from Antara Chemicals) will increase up to seven-fold the "ΔO.D. 8-minute" effect in a taster's saliva.

Cupric chloride ($CuCl_2 \cdot 2H_2O$ cryst. A.R. from Mallinkrodt Chemical Works) in 1.6×10^{-4} M concentration will restore and double the "ΔO.D. 8-minute" effect that was previously removed by electrolytic desalting. If sodium thiocyanate (NaSCN cryst., A.R. from Mallinkrodt Chemical Works) in 7.5×10^{-5} M concentration, which in itself is ineffective, is used in addition to cupric chloride, the effect can be tripled.

These and other experimental findings (to be published) lead us to believe that: (1) taste thresholds for PROP are linked to salivary thyroid activity and (2) this activity is increased in the saliva of "non-tasters" as opposed to that of "tasters."

One of the many ramifications of these findings is that subjects with low thresholds, that is, sensitive "tasters" of the bitter compounds 6-n-propylthiouracil and quinine, dislike more foods as a group than do subjects with high taste thresholds, that is, "non-tasters" of the same two bitter compounds[6].

These relationships suggest the important role of thyroxine and its precursors not only in the mechanism of taste in general and "taste-blindness" in particular but in the selection and rejection of foods, thereby influencing many aspects of constitutional pathology, from dental caries to resistance toward stress.

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GENETIC TUMORS OF NICOTIANA ASSOCIATED WITH CHROMOSOME LOSS

L. G. Burk and T. C. Tso*

GENETIC TUMORS

Figure 8

On the left are tumors, resembling the "Pseudo" type, on a seedling, and on the right are small, newly formed tumors on roots of plants grown in solution culture.

CERTAIN *Nicotiana* hybrids produce teratoid proliferations of a genetic origin. Species within the section *Alatae*, which have nine or 10 haploid chromosomes, produce tumorous hybrids when combined with certain species in other subgeneric sections[1, 2, 3]. A reliable tumor-producing hybrid (*Nicotiana glauca* $n = 12 \times$ *N. langsdorffii* $n = 9$) was used in this study (Figure 8). Plants were grown in soil and in nutrient culture. Solution culture permitted close observation of tumor development through all growth phases, and the tumors which developed provided ideal material for cytological study. Our cyto-technique involved the pretreatment of normal and tumorous tissue in a 0.4 percent colchicine solution for two hours, softening in 1N HCl for two to five minutes, and staining in one percent aceto-orcein.

In smear preparations of tumor tissue, differentiated cells were distorted. The branched spiral elements illustrate the level of distortion and extent of differ-

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entiation (Figure 9*A* and *B*). The tumor is fundamentally a parasitic growth with a metabolic pattern unlike that of the host plant. It is capable of continuous proliferation but of only limited differentiation. The differentiation is characterized by tortuous proto-xylary elements resembling the primary body. Tumors were observed on seedlings (Figure 8), and on all meristems other than the primary growth of the shoot and root-tip.

Chromosome numbers of 17, 18, 19, 36, and 38 were observed in metaphase configurations of tumor cells. A detailed study revealed that the normal chromosome number could only be found in cells of leaf primordia and root-tips (Figure 9*C*). A few root cells in the region of elongation had 19 rather than 21 chromosomes (Figure 9*D*). We feel that such chromosome-deficient cells might serve as tumor initials.

Subsequent counts of chromosomes in tumor cells verified chromosome loss as the probable cause of tumor development (Table I). In well-developed tumors, few division figures could be found. Most of the cells were parenchymatous, some were bi-nucleate, and a number exhibited atypical differentiation. However, in new tumorous growth induced by wounding, and in small root tumors, the proportion of dividing cells was somewhat higher.

Several morphological types were recognized among the larger growths. For convenience, two specific tumors were designated, on the basis of their appearance, as "Pseudo" and "Cauliflower" (Table I). The "Pseudo" form had a characteristic chromosome number of 19, produced leaf-like structures, and had relatively few trichomes. The "Cauliflower" type produced only small shoot-like protuberances a few millimeters in length, had a characteristic chromosome number of 18, and produced numerous glandular trichomes. The possibility that two or three specific chromosomes were deleted in the cells which initiated the morphological types described above is suggestive of further research.

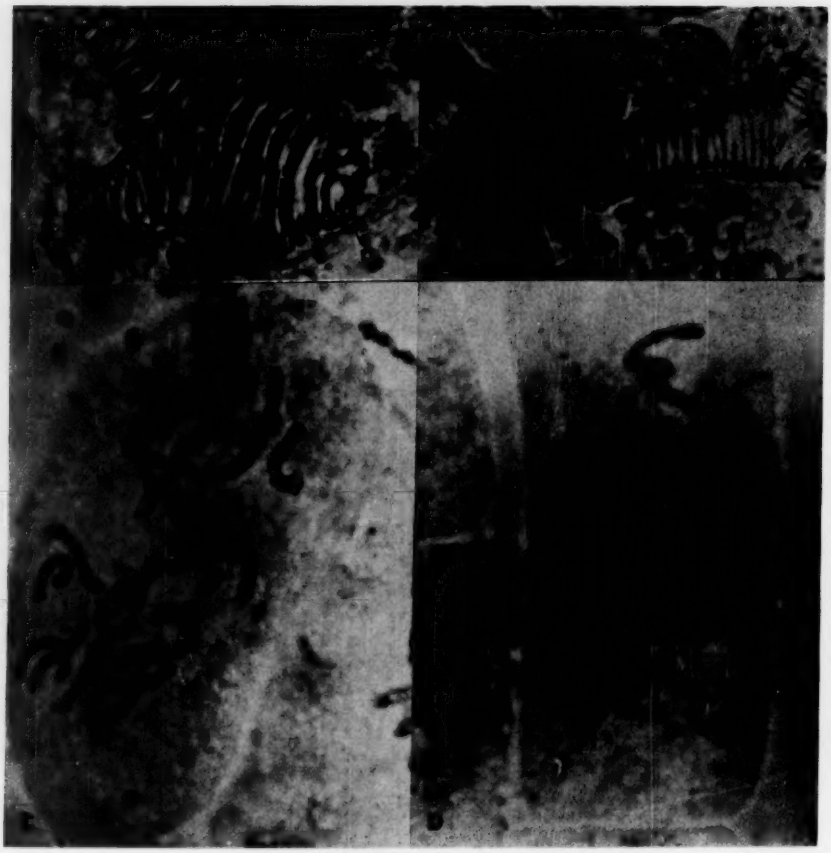
Mixtures of different morphological forms were often observed in very large tumors. On one plant, a group of knob-like tumors occurred on roots exposed at the soil surface. A chromosome number of 38 was observed in three cells and at least 35 chromosomes were present in nine other cells. Because of the limited data available on cells of the knob-like tumor, they have not been included in the table but have received this brief mention because of their apparent polyploidy.

Research on genetic plant tumors has often involved the F_1 hybrid *N. glauca* × *N. langsdorffii* without a corresponding report of chromosome loss. Our material may be unique in this regard, and the tumors examined by other workers may have contained cells with 21 chromosomes. At the time when this paper was under review, we were of the opinion that only Kostoff[2] had reported chromosome numbers in tumor cells. He observed tumor cells with 21, 23, 27, 30, and 42 chromosomes and root-tip cells with 21 and higher aneuploid numbers. A recent report[5] indicates that chromosome numbers in tumor cells of *Picea glauca* vary from three to 70, whereas the majority of normal cells contain the diploid chromosome number.

The current concept of tumor development as a function of aberrant metabolism[1] or disturbed auxin metabolism[4] agrees with our observations of chromosome loss, since the metabolic

TABLE I. Chromosome numbers in cells of normal and tumorous tissues of the F_1 hybrid *N. glauca* × *N. langsdorffii*

	Frequency of cells with indicated chromosome numbers					
Kind of tissue	17	18	19	20	21	36
Normal root-tip			8		33	
Normal leaf-tip					3	
Small root tumors	2	20	25			
"Cauliflower"	1	12				1
"Pseudo"		2	18			
Total	3	34	51		36	1



DISTORTION IN TUMOR TISSUE

Figure 9

A and *B*—Distorted spiral elements. *C*—Root-tip cell with 21 chromosomes. *D*—Root cell with 19 chromosomes.

pattern of cells with 18 or 19 chromosomes cannot be the same as that of cells with the normal number of 21. However, the contention that chromosome aberration or mutation may not be factors in the initiation of tumors[4] does not agree with our observations.

The interspecific hybrid population *N. glauca* × *N. langsdorffii*, which we examined, provided data which indicated that chromosome loss may be the cause rather than the result of genetic plant tumors. Several tumor types were recognized by morphological differences and such characteristic chromosome numbers as 18, 19, and possibly 38, a multiple of 19. No tumor cells with 21 chromosomes or multiples thereof were found. It would appear, therefore, that chromosome loss is an important factor in the transition of a normal cell to an eventual tumorous role.

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"OPEN EYELIDS" IN NEWBORN MICE

JUDITH A. MACKENSEN*

"OPEN EYELIDS," symbol *oe*, is caused by the action of a single, recessive, autosomal gene with complete penetrance. This mutation is similar in its inheritance and expression to "eyelids open at birth"[5] and "eyelids open"[4]. Mice with open eyelids at birth are also produced as part of the syndromes of three other mutations: myelencephalic blebs[3], harelip[6], and congenital hydrocephalus[1].

The first animals with "open eyelids" were found shortly after birth in litters of two female sibs of inbred strain 129/RrSv†, and these animals were the progenitors of the *oe* colony. Breeding pens of the colony are checked each week and those with pregnant females are checked daily (except on weekends) for new litters.

The eyes and eyelids were preserved in Vandegrift's fixative and stained with hematoxylin and eosin for histological examination. The embryos used in this study were aged according to Grüneberg's scale[2].

Genetics

The results of various crosses made to determine the inheritance of "open eyelids" are summarized in Table I. Normal-appearing animals (*Oe-*) were tested for the presence of the *oe* gene by mating them with known heterozygous (*Oeoe*) or affected (*oeoe*) animals. The individual being tested was considered to be a homozygote (*OeOe*) if it produced all normal offspring from a minimum of 15. Outcrosses of affected (*oeoe*) and known heterozygotes (*Oeoe*) were made to strain C3HeB/FeJ and unrelated strain 129 mice (*OeOe*). There was no indication of either a sex difference or of a maternal age influence on the incidence of *oeoe*. All of the *oeoe* animals have been bilaterally affected. The data, in Table I, conform to those expected for a single, recessive, autosomal gene.

Description

Affected animals are readily distinguishable from their normal littermates at birth. The eyelids of newborn animals are wide-open, with inflamed eyeballs showing (Figure 10*A*), and within a few hours the eyes are covered by scabs joining the upper and lower eyelids. These scabs are lost by the time the eyelids normally open; the eyes then appear somewhat redder than is normal for pink-eyed mice. The expression of the gene varies in adult animals. Usually, most of the cornea is opaque (Figure 10*B*), but in some animals there is only a small area of faint opacity. A corneal staphyloma is present in the more extreme cases. Adult *oeoe* animals are usually microphthalmic.

Homozygous (*oeoe*) embryos cannot be distinguished grossly from their normal littermates until the seventeenth day, when the eyelids normally close. Fourteen eyes of 16- and 17-day-old embryos, from matings of *oeoe* × *oeoe* and *Oeoe* × *oeoe*, were sectioned and examined histologically. In eight eyes, a slight protrusion of the lens into the anterior chamber was accompanied by a slight reduction in the width of the cor-

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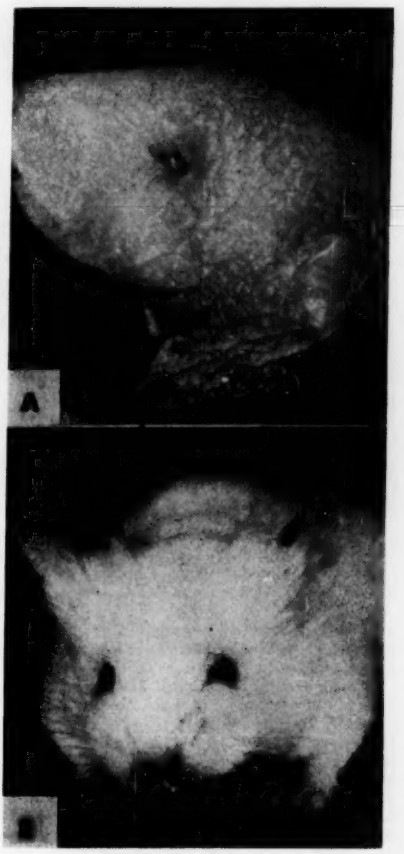
†Standard symbols for the inbred strains are in accordance with the rules and standard symbols given in "Standardized nomenclature for inbred strains of mice, 2nd listing," prepared by the Committee on Standardized Genetic Nomenclature for Mice, published in *Cancer Research*, Volume 20, February. 1960.

nea, which was, presumably, stretched over the protruding lens. Three of these also had abnormal, folded retinas (Figure 11*A*). Lens protrusion without corneal reduction was present in four eyes, one of which also had a folded retina. The other two eyes appeared normal. In nine eyes of 14- and 15-day embryos, from matings of *ococ* × *ococ*, this retinal folding was not present, and the eyes appeared essentially normal with only a very slight protrusion of the lens in four of them.

The retina was not folded in eight eyes of animals ranging in age from term embryos to 10-month-old adults. Protrusion of the lens was present in young animals, but not in the adult eyes examined. The cornea was constricted in 18-day embryos and newborn animals, but it was somewhat thickened in older animals. The staphylomas were composed of a nipple-like structure of fibrous tissue protruding from the cornea. The eyelids appeared to be both grossly and histologically normal.



MANIFESTATIONS OF "OPEN EYELIDS"

Figure 10

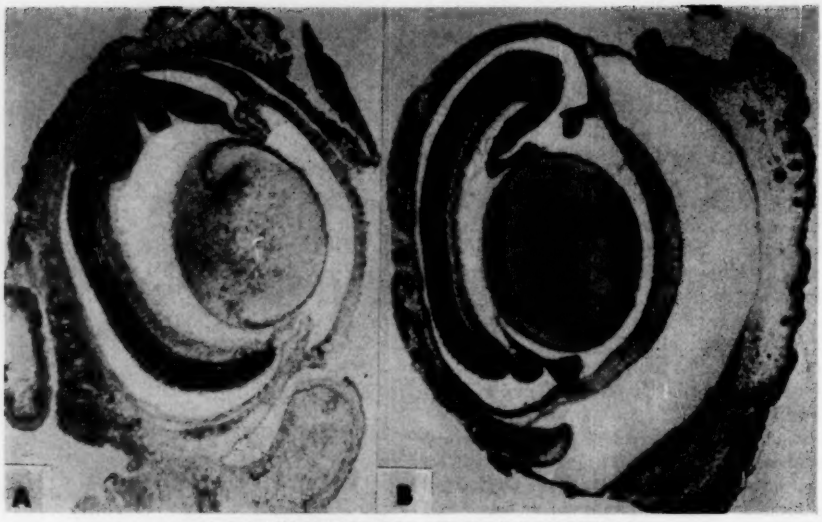
A—Newborn *ococ* animal, showing eyelids open and exposed eyeball. *B*—Adult *ococ* mouse. Note opaque cornea in the left eye and nearly normal right eye.

Discussion

A recessive mutation, "open eyelids," occurred spontaneously in inbred strain 129. Mice with the eyelids open at birth have been found associated with three other mutations: harelip (*hphp*)[6], congenital hydrocephalus (*chch*)[1], and myelencephalic blebs (*mymy*)[3]. In the case of congenital hydrocephalus, Grüneberg believed that stretching of the skin over the protruding cranium prevented eyelid closure[1]. The blebs of *mymy* embryos mechanically interfered with eye development, resulting in disorganized retinal and choroid layers, reduction in the size of the eye, defective or missing eyelids, and opacity of the lens and cornea[3]. The retina was folded in several *ococ* embryos, and in young animals the lens consistently protruded somewhat into the anterior chamber of the eye, while the cornea appeared constricted. The eyes of adults were usually

TABLE I. Genetic ratios obtained from breeding affected *(ococ)*, heterozygous *(Ococ)*, and normal *(OcOc)*

Hypothetical genotype of parents ♀ × ♂	No. born	No. *ococ*	% *ococ*	χ^2
Ococ × *Ococ*	726	179	25	(3:1) 0.045
Ococ × *ococ*	755	324	43	(1:1)15.
ococ × *Ococ*	211	98	46	(1:1) 1.066
ococ × *ococ*	95	95	100	
OcOc × *ococ*	149	0	0	
OcOc × *Ococ*	103	0	0	



EYES OF NORMAL AND AFFECTED EMBRYOS
Figure 11

A—Seventeen-day *oeoe* embryo, showing open eyelids, slightly protruding lens, narrow cornea, and folded retina. *B*—Seventeen-day normal embryo.

reduced in size, with thickened, opaque corneas, and normal-appearing retinas, lenses, and eyelids.

"Open eyelids" is similar to two other recessive mutations, "eyelids open at birth" (*oo*)[3, 5] and "eyelids open" (*coco*)[4]. In all three mutations, the eyelids fail to close during the latter part of embryonic life, and the animals are born with the eyeball exposed and subject to injury. A hemorrhagic exudate occurs in all three which usually closes the opening between the lids with the formation of a scab which lasts for several days. The eyes of adults have some opacity of the cornea. Staphyloma and microphthalmia occur in both *oo*[3] and *oeoe* animals, but are not mentioned in the case of *coco*[4]. There are, however, some differences among these mutations. "Eyelids open at birth"[5] and "eyelids open"[4] animals are affected both bilaterally and unilaterally, whereas "open eyelid" animals have been affected bilaterally only. "Eyelids open" affects males twice as frequently as it does females, and its penetrance, in both sexes, declines with litter seriation[4]. Neither of these effects is found in "open eyelids."

It is possible that these three similar mutations are the same or alleles. The manifestations are similar, and the genetic background on which the gene is acting could produce the differences noted, i.e., differences in sex, laterality, penetrance, and maternal influence. In the absence of breeding tests, the question of identity or allelism cannot be settled conclusively.

Summary

The effects of a mutation in the mouse, named "open eyelids," symbol *oe*, and its similarity to two other mutations is described. The condition is caused by the action of a single, recessive, autosomal gene with regular manifestation. Affected animals have the eyes exposed from birth and subject to injury. Microphthalmia and corneal opacity and staphylomas are present in the adults. Folding of the retina, slight outward protrusion of the lens, and constriction of the cornea occur in *oeoe* embryos.

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A HERITABLE PARTIALLY MALE-STERILE CHARACTER IN COTTON

NORMAN JUSTUS AND C. L. LEINWEBER*

A heritable form of partial male sterility has been found in Upland cotton. Brown† identified the original partially male-sterile plant as a monosomic. No asynapsis was found in the parent plant. Some of the leaves of the original plant tended to be less deeply incised than normal cotton leaves. Further, the parent and sterile segregates had small flowers and the terminal leaves of the mature plants were rugose.

Materials and Methods

The original partially male-sterile parent plant was found in a plot of Western Stormproof Upland cotton at Paradise, Oklahoma, in 1955 by the senior author[1]. This plant was moved to the greenhouse and several grafts were made from it. It was outcrossed to fertile Upland plants as both a male and female parent, and F_2 and first backcross to the partially male-sterile populations were produced.

The segregating generations were scored for percent sterility and fertility.

TABLE I. Sterility scores of individual flowers of 10 randomly chosen field-grown *ms*-1 *ms*-1 Upland cotton plants

	Flowers with indicated sterility score*				
Plant	0	1	2	3	4
1	2	..	1	..	1
2	2	4	..	..	..
3	..	..	..	2	2
4	5	..	..	..	..
5	1	..	1	1	1
6	4	..	..	..	..
7	7	..	..	..	..
8	5	..	1	..	..
9	0	3	..	..	..
10	5	4	1	..	..

*0 = No anthers dehisced.
1 = About 25% of anthers dehisced.
2 = About 50% of anthers dehisced.
3 = About 75% of anthers dehisced.
4 = All anthers dehisced.

TABLE II. The χ^2 analysis of F_2 and backcross populations of reciprocal crosses of partially male-sterile and normal plants of Upland cotton

Population	Number of plants		Segregation ratio	χ^2	P
	Fertile	Sterile			
F_2:					
ms-1 *ms*-1 × normal	327	117	3:1	.432	.70-.50
normal × *ms*-1 *ms*-1	379	145	3:1	1.995	.20-.10
Total	706	262	3:1	2.427	
Deviation				2.204	.20-.10
Heterogeneity				.223	.70-.50
BC_1:					
(*ms*-1 *ms*-1 × normal) × *ms*-1 and (normal × *ms*-1 *ms*-1) × *ms*-1 *ms*-1	89	73	1:1	1.580	.30-.20

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The scale used to score the plants is delineated in Table I.

Flowers on each plant were examined a number of times during the season, and the score recorded on a paper tag tied on each plant. If a plant had less than 100 percent anther dehiscence at any scoring time during the season, it was considered sterile. Limited progeny testing indicated this method was sound.

Results and Discussion

Segregation results from two F_2 populations and a BC_1 population are presented in Table II. Assuming that partial male sterility is due to one recessive gene, the value of P for the chi-square values ranged from .20-.10 to .70-.50.

The heterogeneity test, Table II, indicated no significant difference between populations when the partially male-sterile parent was used as the male or female. The sterility factor is easily transferred through either the pollen or the ovules. It is not likely that the monosomic condition is responsible for the sterility expression. If it were, 3:1 and 1:1 would not be the ratios in the F_2 and BC_1 generations. In the F_2 the possible nullisomic probably would not survive. Also, if the monosomic were responsible, the F_1 should have segregated.

The sterility expression was modified by environmental conditions. The data in Table I show that the sterility score of the same plant varied during the season; however, in most cases the shift in degree of sterility was not radical. Further, a much higher rate of pollen dehiscence was observed, on both "stubbed" material and seedlings, in the greenhouse than in the field. Therefore, self-pollinated seeds are obtained rather easily in the greenhouse.

If a field environment could be found that would produce a degree of anther dehiscence equal to that of the greenhouse, then commercial hybrid cotton seed production would be feasible through large-scale production of homozygous partially male-sterile seed. To this end homozygous partially male-sterile siblings were planted at Stillwater, Oklahoma; Chickasha, Oklahoma; Stoneville, Mississippi; and Iguala, Mexico. There was no observable difference in percent pollen shed at any of these locations. The F_1 generation was always fully fertile.

By utilizing this gene in a backcross population a limited amount of hybrid seed could be produced for breeding purposes. Marker genes would fit into such a scheme especially well.

The gene symbol *ms*-1 is proposed for this character.

Seed from this material has been placed in the Upland Cotton Regional Collection at Stoneville, Mississippi.

Summary

A recessive gene for partial male sterility has been found in Upland cotton. The sterility expression is modified by environment but not radically. It is suggested the gene symbol *ms*-1 be used for this factor. Seed of the genotype *ms*-1 *ms*-1 has been placed in the Upland Cotton Regional Collection at Stoneville, Mississippi.

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